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ARCHIVES OF OTOLARYNGOLOGY

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OTOLOGIC OBSERVATIONS IN TRAUMA OF THE HEAD

A CLINICAL STUDY BASED ON FORTY-TWO CASES *

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MILWAUKEE

Injuries to the head should be of peculiar interest to the otologist, because in so many one of the divisions of the ear apparatus is affected. As early as 1905, Passow [1] stated that in severe injuries to the skull, the ear practically never remains intact. Often only a mild force of impact produces damage in the labyrinth. In just how large a proportion of cases of injury to the head, ear symptoms develop it is difficult to determine, as the patients are usually first seen by the general physician or surgeon, and are only referred to the otologist for examination at some later date if ear trouble is suspected. However, Mygind [2] found otoscopic changes in 50 per cent of 142 patients with injuries of the head, consecutively examined, and traumatic vestibular disease in 33.3 per cent. About two thirds of the patients were examined before the lapse of the first week, and many of them only a few hours after the injury.

Careful and thorough examinations must be made in all cases of this sort as soon after the injury as possible, and again at regular intervals over a considerable period. It is too easy to make a superficial examination, which, as Kobrak [3] pointed out, gives only the poorest picture of the real, deep, underlying changes present, and which helps to put these patients into the class of malingerers or of persons with psycho-neuroses.

The severity of the injury apparently does not bear any direct relation to the development of cochlear and vestibular symptoms. Mygind found concussion of the brain in only a little more than 50 per cent of all patients examined, and did not find any direct relation between this fact and the otologic symptoms. In my own series of patients, unconsciousness was noted in the history twenty-nine times; in nine, consciousness was not lost; in four records, loss of consciousness was not noted.

There seems to be a great deal of confusion in the literature as to just what is meant by concussion of the brain, Gehirnerschütterung or

* Candidate's thesis for American Laryngological, Rhinological and Otological Society.

1. Passow, A.: Die Verletzungen des Gehörorgans, Wiesbaden, 1905.

2. Mygind, S. H.: Traumatic Vestibular Diseases, Acta oto-laryng. **1**:515, 1919; Vestibular Examinations in Normal Individuals, ibid. **1**:527, 1919.

3. Jansen and Kobrak: Praktische Ohrenheilkunde für Aerzte, Berlin, Julius Springer, 1918, p. 338.

commotio cerebri. According to Miller,[4] there is an increasing tendency to limit the term concussion to those cases in which the clinical syndrome of cerebral compression is not present. The pathologic definition, widely taught and accepted, is that concussion is part of a condition which has for its pathologic basis scattered microscopic contusions and petechial hemorrhages. According to Miller, a physiologic definition of concussion would be a group of symptoms which are the result of a temporary inhibition of one or more of the medullary centers, but which are not accompanied by pathologic lesions.

While it is undoubtedly true that there are well established cases of death from pure concussion, the weight of opinion seems to be that in the great majority of these fatal cases there is a mixture of concussion, contusion and hemorrhage.

Pure concussion does not mean cell death, but rather cell paralysis, and complete recovery and restoration of function should take place. Following contusions and petechial hemorrhages, however, there are inflammatory reactions and processes of repair, and here sequelae of a more or less permanent character may ensue. All these facts have an important bearing on the problem, because, as Kobrak [3] said, in every injury of the head of sufficient intensity to produce a commotio cerebri the possibility of an associated commotio labyrinthi must be considered.

According to Stenger,[5] the more often one examines patients with so-called traumatic neurosis occurring as the result of injuries to the head, the more often one finds a more or less constant symptom-complex consisting of headache, vertigo, tinnitus, partial or complete deafness in one or both ears, feeling of weakness, etc. These symptoms are characteristic and point so definitely to the labyrinth that even after their recession the more general nervous manifestations appearing later can be brought into direct connection with the original picture of the disease. Stenger did not regard these general nervous manifestations, such as headache, tinnitus, vertigo, inability to concentrate, ataxia and sensibility disturbances, as the result of a general neurosis, but rather referred them to a condition of the labyrinth from which the patient had recovered. In some of the cases the vertigo, in others the hearing defects and in still others the general nervous manifestations are more prominent. Stenger used the term "traumatic neurosis" for those cases in which evidence of organic disease is not present. If symptoms are found arising from the labyrinth, then the resulting general nervous manifestations must be referred to an injury of this organ, and for these cases he reserved the designation "traumatic labyrinth neurosis."

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4. Miller, G. G.: Cerebral Concussion, Arch. Surg. **14**:891 (April) 1927.

5. Stenger: Ueber die Arten der nach Kopfverletzungen auftretenden Neurosen; Die traumatische Labyrinthneurose, Deutsche med. Wchnschr. **31**:63, 1905.

THE CHARACTER AND MECHANICS OF THE INJURY

In general, all injuries to the head are caused by some broadly acting force which has a wide point of impact against the skull. Either the skull is struck a blow, or it is set into rapid motion by a fall and suddenly comes to rest against some broad, solid substance. Thirteen of my cases were due to automobile accidents; fourteen to falling objects striking the head; ten to a fall from some height; two to the head being struck a blow; one to the head being jammed between the closing doors of a street car, and one to the head being jammed between a conveyer chain and a sprocket wheel.

Just what takes place mechanically in these injuries? F. Breslauer-Schück [6] called attention to three phases in the original injury of the head:

1. At the moment of impact, the skull strikes against the brain, because the brain, floating free in its liquor, lags behind.

2. In the next phase, the brain is taken along with the skull in the direction in which the skull is moving.

3. In the third phase, the side movement of the skull is stopped by the vertebrae or by its striking against some solid object and coming to rest. The brain is still moving toward this point of rest, and it now strikes the opposite surface of the skull.

Le Count and Apfelbach [7] also called attention to this mechanism of injury and said that "the injuries of the brain concomitant with fractures of the cranial bones are preponderantly of the outside of the brain, owing partly to the inbending of the cranial bones but chiefly to bumping of the brain against the cranial bones."

An injury, as outlined previously, will produce damage to the skull itself, and also to the contents of the skull, among which must be considered the brain, the cerebrospinal fluid and the vascular system of the blood (Brunner [8]).

With regard to the injury to the skull itself, Passow [1] made the statement that Bruns was the first to describe the elasticity of the adult skull, while Barnick [9] called attention to the theory of Von Wahl and Messerer which is based on these elastic properties of the skull. According to this theory, all skull fractures which are caused by a blunt force

6. Breslauer-Schück, F.: Physiologische Betrachtungen zur Lehre der Gehirnerschütterungen, Beitr. z. klin. Chir. **121**:590, 1921.

7. Le Count, E. R., and Apfelbach, C. W.: Pathological Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.

8. Brunner, Hans: Pathologie und Klinik der Erkrankungen des inneren Ohres nach Stumpfen Schädeltraumen, Monatschr. f. Ohrenh. **59**:697, 1925.

9. Barnick, Otto: Ueber Brüche des schädelgrundes und die durch sie bedingten Blutungen in das Ohrlabyrinth, Arch. f. Ohrenh. **43**:23, 1897.

are divided into bending fractures (Biegungsbrüche) and bursting fractures (Berstungsbrüche). A small force applied to a circumscribed area of the skull will produce the bending type of fracture in its immediate neighborhood. The bursting or explosive type of fracture is caused by a broadly acting compressing force exerted over a large part of the skull capsule, and these fractures find their greatest expression at the base. Schönbauer and Brunner [10] pointed out that, with a broadly acting force, the skull is compressed in the direction of the acting force and is spread in a direction at right angles to this force producing the bursting type of fracture. This type finds its greatest expression at the base of the skull. Barnick,[9] Stenger,[11] Schönbauer and Brunner [10] and others called attention to the fact that the base of the skull is its weakest portion, consisting, as it does, of strong pillars or buttresses, such as the pyramids and the wings of the sphenoid, with weaker portions between them which are perforated by many foramina, whereas the vault is rather uniformly resistant to any force applied to it. Of the base itself the middle fossa is weaker than the anterior and posterior fossae. Barnick [9] found that the great majority of basal skull fractures affected the middle cranial fossa; Borden [12] found the petrous portion of the temporal bone most often involved in fractures of the base; while Schönbauer and Brunner,[10] studying fractures in fifty-seven macerated skulls, found the middle fossa involved in forty-five, or 80 per cent, as against 60 per cent reported by Matti.

It is seen, therefore, that the temporal bone is frequently damaged directly by the basal skull fracture. Even when it is not fractured itself, Stenger thinks it must be involved in heavy swinging movements and that its contents, consisting of endolymph and membranous organs, must be thrown into violent commotion by the blow, and thereby damaged. What this damage may be can be ascertained only by experimentation on animals, and this I shall take up later.

Schönbauer and Brunner found the temporal bone injured in 45 per cent of the cases which came to autopsy shortly after the accident. They believed that this percentage was too small, because the specimens were examined macroscopically only; and they pointed out that many fine fissures which cannot be seen macroscopically can be made out on microscopic examination.

The fractures of the temporal bone are divided into three varieties, longitudinal fractures, transverse fractures and avulsion of the tip of the

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10. Schönbauer and Brunner: Schadebasis Brüche, in Alexander and Marburg: Neurologie des Ohres, 1927, vol. 2.

11. Stenger: Beitrag zur Kenntniss der nach Kopfverletzungen auftretenden Veränderungen im inneren Ohr, Arch. f. Ohrenh. **79**:43, 1909.

12. Borden, C. R.: Otologic and Rhinologic Complications of Skull Fractures, J. A. M. A. **53**:429 (Aug. 7) 1909.

petrous bone. Among twenty-three fractured petrous bones in the collection of Schönbauer and Brunner there were fourteen longitudinal fractures (61 per cent), three transverse fractures, two compound fractures, one avulsion of the petrous tip and three cases in which the mastoid tip alone was involved. The longitudinal fractures were always combined with fractures of the middle and anterior fossae.

The longitudinal fractures usually run along the anterior border of the pyramid, according to Stenger,[11] Ulrich[13] and Schönbauer and Brunner.[10] They come from the anterior fossa or the region of the Gasserian ganglion, and end in the tegmen antri or tympani and in the external canal. This sort of fracture causes hemorrhage into the external canal, which is of itself diagnostic of fracture of the basal skull, though not necessarily of fracture of the labyrinth, as Ulrich[13] and Brunner[8] point out. The longitudinal fracture passes through the canalis musculotubarius and through the tympanic cavity. The malleus and ambos may be dislocated, and the stapes may be damaged or broken. Through the mobilization of the malleus, the drum is ruptured. As Ulrich[13] points out, if the fracture passes posterior to the malleusambos articulation, it may involve the facial nerve in the area of the knee ganglion. The longitudinal fractures only rarely send off shoots into the bony labyrinth. In my series there were nine cases in which I believe a diagnosis of longitudinal fracture was sustained because of the history of unconsciousness, bleeding from one or both ears and the loss of hearing, especially in the upper tone range.

The transverse fracture of the pyramid is, as has been seen, much less common than the longitudinal fracture. Its course, however, is just as typical. It crosses the pyramid at right angles at its weakest portion, which is the region of the vestibule, and, as a rule, it spares the more compact cochlea, according to most observers, though Ulrich holds that it more usually crosses the cochlear portion. All the membranous portion of the inner ear is destroyed. The fracture may or may not involve the lateral wall of the inner ear and enter the tympanic cavity. If it does so, it may be associated with hemorrhage from the ear; this, however, is extremely rare. The following case record is an example of this type of fracture. It is the only instance of a transverse fracture of the pyramid which I have in my series.

Case 1.—*History.*—The history was taken on April 7, 1927, through an interpreter. Pete Machtika, aged 51, was struck in the area of the left vertex by a falling plank on March 15, 1927. He was unconscious for four or five days. On regaining consciousness he had severe continuous headache and marked continuous dizziness. The vertigo was greatly increased when he attempted to get out of bed. He could not hear with the right ear as soon as he became

13. Ulrich: Klinische und anatomische Untersuchungen über Verletzungen des Gehörorgans bei Schädelbasis frakturen, Schweiz. med. Wchnschr., 1921, p. 24.

conscious. He spat blood for several days after the accident but did not know whether he bled from the ears.

When seen by me he still had headache in the occipital region and pain in the region of his wound. He complained of tinnitus in his right ear. He stated that on arising in the morning or on looking up suddenly dark spots appeared before the eyes, but he did not get dizzy at that time. He could not hear with his right ear.

Examination.—Spontaneous Phenomena: After bending his head forward several times the patient stated that he was dizzy and had a sense of rotation. The Rhomberg reaction was negative. In his forward gait with the eyes closed he deviated to the right. There was no spontaneous past pointing with either arm, but there was a spontaneous deviation of the left arm to the left. Deviation with the right arm was not noted. A spontaneous rotary-horizontal nystagmus was present to the left side only.

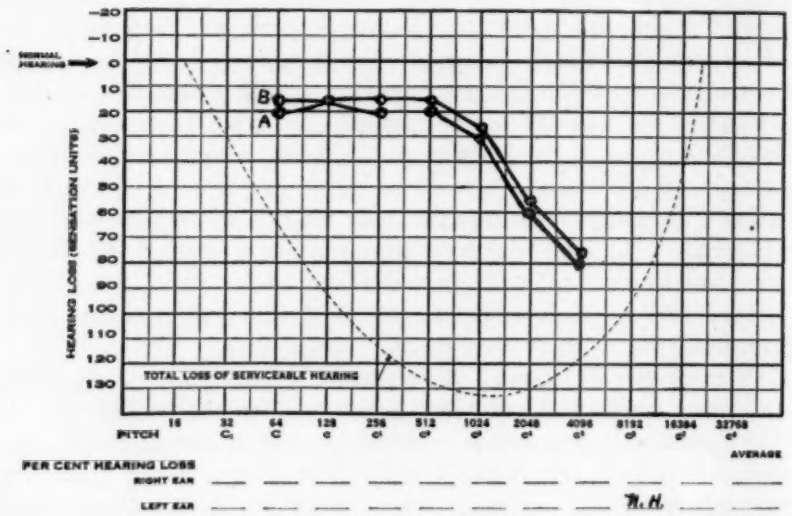

Chart 1 (case 1).—Audiograms of left ear: *A*, taken on April 6, 1927; *B*, on July 14, 1927.

Eyes: Both pupils were equal and reacted to light. The ocular muscles were not paralyzed. There was not any facial paralysis.

Ears: Both external canals were clear. Both drums were normal, except for slight retraction. There were not any scars or perforations.

Hearing: The Weber reaction was referred to the left. Bone conduction on the right side was referred to the left. The Rinne reaction on the left side was positive and bone conduction on the left was less than eight seconds. With noise apparatus in the left ear he heard nothing with the right. A *C* 256 fork was heard 4 inches (10.16 cm.) from left ear. This fork was connected to a rubber tube the other end of which was connected to the right external meatus by an ear piece; when it was struck, it was not heard until it was moved within the 4 inch radius of the left ear; then it was heard immediately. The upper tonal range of the left ear was also considerably damaged as may be seen in the audiogram, chart 1.

Vestibular Tests.—Right Ear: A large amount of water at 15 C. did not produce nystagmus. The spontaneous nystagmus to the left remained unchanged. Vertigo was not present but there was slight past pointing to the right.

Left Ear: Fifteen cubic centimeters of water at 15 C. produced in thirty seconds a rotary-horizontal nystagmus to the right, on the forward look, which lasted about sixty seconds. There was past pointing to the left with the right arm but not with the left arm. Vertigo was not noted.

Turning Tests.—Ten turns to the right in twenty seconds produced a nystagmus to the left of eleven seconds' duration. Ten turns to the left in twenty seconds produced a nystagmus to the right of seven seconds' duration.

Second Examination.—On July 14, 1927, the patient complained of some headache on the right side which was more or less constant. He said he was dizzy when he bent down but he did not describe this as a rotation sensation.

Spontaneous nystagmus was not present. There was spontaneous past pointing to the right with the right arm but none with the left arm. The right ear was entirely deaf. The Rinne reaction was positive on the left. The reaction to the Schwabach test was only slightly shortened. A watch was not heard on contact. After ten turns to the right the after nystagmus was nine seconds and after ten turns to the left it was eight seconds.

Diagnosis.—A diagnosis was made of transverse fracture of the right pyramid, with hemorrhage into the left labyrinth.

Avulsion of the tip of the petrous temporal bone is relatively rare and should not affect the inner ear. It may, however, cause injury to the nerves at their entrance into the internal auditory meatus.

In addition to the damage suffered by the skull capsule itself, or even without any damage to the skull, its contents may be seriously injured. Among these must be considered the brain, the cerebrospinal fluid and the vascular system of the blood. Changes to these structures are probably brought about in two ways: first, as Le Count and Apfelbach pointed out, the brain, because of its suspension in its liquor, strikes against the skull at the point of impact of the force at the beginning of the head movement and against the directly opposite portion of the skull when the head movement is checked. In these areas pial hemorrhages occur, and the brain itself is undoubtedly contussed; a second factor in the injury to the brain tissue, as Brunner pointed out, is the change in form of the elastic skull, with its resultant compression of the brain.

As the form of the elastic skull is changed and the brain itself is compressed, the lateral ventricles are also compressed and tend to collapse. Their cerebrospinal fluid is forced through the foramen of Munro into the third ventricle, and from there through the aqueduct of Sylvius into the fourth ventricle. As the fluid cannot escape further it sets up a whirlpool action here whose force is exhibited against the walls of the fourth ventricle, and particularly against its floor. Close under the surface of the floor are located the nuclei of the cochlear and vestibular branches of the eighth nerve, and these can easily be injured by the pressure wave of fluid.

The third element of damage to the contents of the skull is the trauma to the blood vessels and their regulating mechanism. Ricker [14] and Knauer and Enderlen [15] came to the conclusion, from animal experimentation, that a state of traumatic paralysis of the vasoconstrictor nerves arises after the trauma which Knauer and Enderlen think is due to trauma to the midbrain. Ricker was able to show after trauma to the brain that areas of the brain are found in which the circulation is in a condition of stasis or of prestasis. In this condition, the vasoconstrictors are paralyzed and nonirritable. The end-result of stasis, as far as tissue is concerned, is an interruption of tissue function, and in the end a necrosis of the tissue itself.

If stasis develops suddenly, over a large territory, diapedesis does not occur; but if it develops gradually, and if one capillary region is in a condition of stasis, while a neighboring one is in a condition of prestasis, a great amount of diapedesis can take place, and this condition, which lasts until widespread stasis appears, can lead to the widespread formation of infarcts.

If the stasis does not last long enough to destroy the brain substance in toto, then Ricker held that the parenchyma is affected before the stroma, and to the parenchyma belongs the neuroglia. Degeneration changes then take place in this tissue. Phelps [16] also called attention to the effect of the blow on the vasomotor center causing a vasomotor depression of the cerebral vessels, with paralytic dilatation of the capillaries, stasis, thrombosis and punctate extravasation, which is increased by the edema of the brain.

According to Brunner,[8] the force exerted against the outside of the skull, which compresses its contents, the brain and cerebrospinal fluid, and damages the vascular control, is also exerted against the inner walls of the skull and, thus, against the temporal bone. This wave of pressure does not have any effect against the temporal bone itself, but it does affect the saccus endolymphaticus lying on the posterior surface of the pyramid and also the internal auditory meatus. As a result of the pressure on these two structures a wavelike motion of the perilymph and endolymph arises, which is directed from behind and above, forward and downward, to break itself at the tip of the cochlea as the lowest most anterior point. Here a whirlpool action of the fluids of the inner ear occurs. The energy of this wave motion expends itself particularly in

14. Ricker, G.: Die Entstehung der pathologisch-anatomischen Befunde nach Hirnerschütterung im Abhängigkeit vom Gefässnervensystems des Hirns, Virchows Arch. f. path. Anat. **226**:180, 1919.

15. Knauer, A., and Enderlen, E.: Die Pathologische Physiologie der Hirnerschütterung, J. f. Psychol. u. Neurol. **29**:1, 1922.

16. Phelps, Charles: An Analytical and Statistical Review of One Thousand Cases of Head Injuries, Ann. Surg. **49**:449, 1902.

the region of the round window, which is the weakest point in the bony inner ear. This accounts for the hemorrhages in this vicinity, and for the bulging of the round window membrane into the middle ear and its occasional rupture.

In addition to the changes produced by this wave motion of the fluid there are circulatory changes in the inner ear, produced partly mechanically and partly by changes in the vasomotor center, in the sense of Ricker and of Knauer and Enderlen.

Trauma of the head may also damage the skull contents in two other ways. It may cause hemorrhage by direct laceration of blood vessels, and it may, because of the movement of the brain within the skull, cause laceration and tearing of cranial nerves. The first of these two factors does not concern otologists so vitally, but the second is of great importance because the eighth is one of the frequently injured nerves. The cranial nerves, according to Ulrich,[17] are injured either by being cut, torn or sectioned by fragments of bone, or by traction. Ulrich found that a nerve is injured by stretching in inverse proportion to its length; hence, a long caudal nerve like the vagus is rarely injured in this way. The auditory nerve, however, is short from its exit from the internal auditory meatus to its entrance into the brain stem, and this probably accounts for the fact that it is the most frequently injured nerve in fractures of the base of the skull.

PATHOLOGIC CHANGES IN HUMAN BEINGS

The otologic symptomatology of injuries of the head is hard to understand unless one has some conception of the pathologic changes produced by the trauma. In the case of severe injuries which cause death, this knowledge can be obtained at the autopsy table. Knowledge so obtained is, however, not sufficient, for, as Brunner [8] pointed out, the pathologic changes in patients who recover may be very different from those in patients who succumb to their injuries. For this purpose animal experimentation must be resorted to. I have the good fortune, furthermore, of having autopsy reports of a few patients who died from some intercurrent condition many years after the injury to the head.

As I do not have any pathologic material of my own, I shall draw on the literature to cover this portion of my work, and I shall review first the pathologic observations in the patients who died soon after injury.

In 1897, Barnick reported the examination of the temporal bones of four patients who died of basal skull fractures. His observations were as follows:

17. Ulrich: Ueber Vagus, Fazialis- und Akusticusverletzungen, Schweiz. med. Wchnschr., 1922, p. 21.

1. In all the cases there were hemorrhages in the narrow nerve canals leading to the otolith apparatus of the saccule and utricle, and to the ampulae of the semicircular canals.

2. Massive hemorrhages were seen in the vascular territory of the maculae.

3. Regular bleeding occurred in the perilymph spaces.

4. Exudations of blood into the endolymph spaces were rarely seen.

5. Massive hemorrhages occurred in the tympanic scalae of the basal coil of the cochlea. Barnick thought that the loss of hearing for the high tones in patients who recovered was due to the injuries in the basal coil of the cochlea.

Sakai [18] studied ten temporal bones shortly after death from skull fractures and reported the following observations:

1. The bony labyrinthine capsule was not fractured in any case.

2. Hemorrhage occurred at the region of the round window membrane eight times and at the oval window eight times.

3. The ligamentum annulare was torn only once.

4. The acoustic nerve and its branches showed marked hemorrhage in all cases. Of the two main trunks, the cochlear nerve showed a rupture of its fibers eight times and the vestibular nerve four times.

5. There were hemorrhages into the soft parts of the utricle, saccule and canals five times.

6. Hemorrhage into the facial nerve occurred once.

7. Hemorrhage into the fallopian canal was seen four times.

8. Hemorrhage into the ligamentum spirale occurred twice.

9. Hemorrhage into the ganglion spirale was observed twice.

Thus, in these specimens, the most frequent injuries to the soft parts of the inner ear were changes in the eighth nerve and its branches and hemorrhages in the region of the round and oval windows. Sakai believed that similar changes in patients who recover may result in (1) a degenerative atrophy of the soft parts of the labyrinth and its nervous elements, and (2) the formation of new bone inside the labyrinth, as in one case of Manasse and one of Nager.

Lange,[19] presenting the examination of the petrous bones of fourteen patients who died of fracture of the skull, called attention to the following facts:

1. Damage to the bony labyrinthine capsule was infrequent.

2. The tegmen tympani or antri was fractured in every case.

3. The tympanic membrane and external canal walls were often injured.

4. The oval and round windows were always intact.

5. There was always more or less blood in the depth of the porus acusticus internus.

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18. Sakai, K.: Anatomische Befunde am menschlichen Gehörorgan nach Basisfraktur, Arch. f. Ohrenh. **85**:188, 1911.

19. Lange, W.: Anatomischer Befund am Gehörorgan nach Basisfraktur, Ztschr. f. Ohrenh. **53**:37, 1906; Beiträge zur pathologischen Anatomie der Verletzungen des Gehörorganes, Passow u. Schaeffer Beitr. **18**:277, 1922.

6. Blood was found in the nerve canals, particularly in the branches of the vestibular nerve.

7. Complete tearing of the nervus acusticus was never seen, but separation of its fibers by blood extravasations was made out.

8. Damage to the facial nerve was not observed.

Ulrich [20] studied microscopically eighteen temporal bones of patients dying from basal skull fractures. In seventeen the line of fracture was parallel to the pyramid. Most of the fractures were along the anterior edge of the pyramid and involved the roof of the middle ear. The middle ear is always injured by this type of fracture. The vestibular nerve is most easily and often damaged, the cochlear nerve next and the facial nerve least. The vestibular end organ was rarely injured. On the contrary, there were signs of injury in the cochlea in all eighteen cases, the most common observation being hemorrhage.

1. All the hemorrhages are in the perilymph spaces, never in the endolymph spaces.

2. The origin of the hemorrhage is always in the scala tympani, and usually in the vicinity of the round window.

3. The tympanic membrane is ruptured only in cases in which the margo tympani is involved.

4. The farther from the ear the fracture lesion is, the more insignificant are the ear lesions.

5. Direct damage to the facial nerve in these longitudinal fractures of the pyramid is not common.

Ulrich said that in true labyrinth fractures the line of fracture is at right angles to the pyramid, and in this type of fracture the cochlea is the most vulnerable part of the labyrinth. Here hemorrhages into the endolymph can take place. In this type of fracture the only portion of the middle ear which can be damaged is its mesial wall. He never found the drum ruptured in this kind of case. The presence of blood in the external canal of itself speaks against a true transverse fracture of the labyrinth. The facial nerve is involved in about one half of all these cases.

Voss [21] recently reported an interesting histologic study of thirty temporal bones of infants who died soon after birth, presumably from pressure. In all of these he found a marked widening and tense filling of the blood vessels of the entire organ of hearing. These extended from the drum over the middle and internal ear, the bony spongiosa, the facial and carotid canals and the porus acusticus internus to the dura

20. Ulrich, K.: Verletzungen des Gehörorgans bei Schädelbasisfracturen, suppl. to Acta oto-laryng. **6**:1, 1926.

21. Voss, O.: Klinische und pathologische-anatomische Folgeerscheinungen geburtstraumatischer Schädigungen des Felsenbein, Monatschr. f. Kinderh. **34**: 568, 1926.

of all three pyramid surfaces. There were, furthermore, microscopic and macroscopic exudations of blood, particularly in the basal coil of the scala tympani.

So it is seen that in human beings dying shortly after injury, the salient pathologic feature is hemorrhage. The hemorrhages are perilymphatic unless the bony labyrinthine capsule is fractured, in which case they can also be in the endolymph spaces. The nerves can be torn before their entrance into the pyramid, in the porus acusticus internus or in the narrow bone canals leading to the end organs in the cochlea and vestibule. What the pathologic end-results of these lesions on the organ of hearing might be one would have to conjecture were it not for the fact that records have been published of examinations of temporal bones in persons who died at more or less remote periods after injuries of the head, and that the effects of milder injuries to the head, which do not produce death, have been extensively studied in experimental animals.

I will take up first the cases of Alexander,[22] Theodore[23] and Manasse.[24] Alexander studied his case forty-four days after the original injury to the head, Theodore twelve years after and Manasse fifteen years after.

Case 2.—The inner ear of a man who died forty-four days after a gunshot wound in the right parietal region was examined histologically by Alexander. Death resulted from meningo-encephalitis. Clinically the man was unconscious immediately after the injury and then he suffered from severe headache, vertigo, tinnitus, nausea and vomiting. Both tympanic membranes were normal. The hearing acuity was slightly diminished. Bone conduction and the upper tone limits were slightly shortened. The watch test on the bone was negative on the right side and positive on the left side. There was spontaneous horizontal-rotary nystagmus to both sides in the end positions, infrequent and of slight intensity. The reflex irritability of both labyrinths was pathologically increased.

The histologic examination revealed the following:

1. There was a circumscribed loss of sensory cells in the cristae of the ampullae.

2. A narrowing of the cochlear canal through a lowering of the vestibular membrane was observed.

3. There was a loss of the highly organized cells of Corti's organ in the vestibular segment of the cochlea. Corti's papilla was composed of indifferent cells. The impairment of Corti's organ by the traumatism was quite grave in the vestibular segment.

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22. Alexander, G.: Die Histologie der inderekten traumatischen Labyrinthverletzung durch Schädelschuss, Festschr. f. Urbantschitsch, 1918; Zur Frage der pathologische Bedeutung der endolymphatischen Labyrinthblutung, Arch. f. Ohrenh. **59**:13, 1903.

23. Theodore, E.: Beitrag zur Pathologie der Labyrintherschütterung, Ztschr. f. Ohrenh. **61**:299, 1910.

24. Manasse, Paul: Zur pathologischen Anatomie der traumatischen Taubheit, Virchows Arch. f. path. Anat. **189**:188, 1907.

4. Atrophy was observed in the peripheral cochlear nerve going to the vestibular portion of the cochlea and also in the corresponding portion of the spiral ganglion.

5. Corti's organ was injured in only circumscribed areas in the basal coil.

6. The changes in the basal papilla of the upper portion of the basal coil and of the middle convolution were much slighter.

It is therefore seen that in this case of Alexander's degenerative changes took place in the neural tissue of the end-organ in regions in which one would expect to find them when one recalls where the hemorrhages were found in the cases of Barnick, Lange, Sakai and Ulrich.

Even more instructive as to what takes place in the ear of a person who suffers and recovers from a severe injury to the head, which does not particularly involve the skull or the temporal bones, is the following case of Theodore.[23]

CASE 3.—*History.*—R. Engelbricht, aged 42, was struck down on Nov. 12, 1896, by a bicycle, his head striking the pavement. He was unconscious for from twelve to fourteen hours. On awakening, he had severe headache, vertigo and vomiting whenever he raised his head. There was also bleeding from the right ear. In his history it was found that in 1886, ten years previously, he had received another blow on the head after which his hearing was not as good as it had been before.

Examination.—In the right occiput was seen a contused wound with edematous edges. The right mastoid was tender. Fissures could not be felt. There was no vomiting. Marked headache was present, particularly when the patient was fixing on an object. Vertigo was still marked in the recumbent as well as in the sitting or the standing posture. The Romberg sign was negative. On forward gait with eyes closed there was considerable staggering to both sides. There was suggillation behind the right ear which extended to the tip of the right mastoid.

Tests of the hearing gave the following results:

Tests of Hearing	Right Ear	Left Ear
Whisper	No hearing	¾ meter
C fork	No hearing	Heard
C 1 fork	Hearing weak	Heard
Galton whistle	Normal hearing	Heard
Weber test	Referred to right ear	
Bone conduction	Four seconds short	Normal

Eye signs were not present.

By December 5, the swelling of the posterior canal wall had receded. The drum was visible and hemorrhagic. A whisper was heard at 10 cm. by the right ear.

By Jan. 7, 1897, the headaches only recurred after exertion and excitement. The vertigo was entirely gone. The right external canal was normal. The right drum was cloudy and thickened. The hearing for the whisper was 25 cm. on the right and ¾ meter on the left; the *C* 1 fork was heard by both ears; the Weber test was not lateralized. The Rinne reaction was positive on the right side.

In 1908, the patient was readmitted to the hospital because of pulmonary tuberculosis. At this time examination of the ears showed both drums thickened and retracted. Results in the tests were as follows: whisper, not heard by either

ear; conversation, heard by both ears at ear; all forks heard; Galton whistle, right, 2.0 and left, 1.8; Weber test not lateralized; Schwabach test, right, ten seconds short, and left, seven seconds short; nystagmus was not observed.

Death occurred on Feb. 27, 1908.

Section (Professor Chiari).—No signs of skull fracture were present. At the apex of both temporal lobes of the brain, as well as in the left frontal lobe, there were hemorrhagic scars. Fracture or fissures could not be made out in either temporal bone.

Microscopic Examination.—The right petrous bone showed an intact labyrinthine capsule. The tympanic membrane, the ossicles, the round window and its membrane, and the oval window with the ligamentum annulare were all normal. Changes had not occurred in the vestibule or semicircular canals. Changes were found in the cochlea, especially in the basal coil, while the upper coils remained intact. Corti's organ in the basal coil consisted only of a heap of cells the details of which were hard to differentiate. The much thinned inner and outer columns could still be distinguished with their floor cells. The inner hair cells were replaced by round and cubical cells with well defined nuclei, cells which were partly granular or hyaline. Similar changes were also found in the outer hair cells and in Deiters' cells. In some sections the membrana tectoria lay tight up against Corti's organ, so that there was a marked reduction in the lumen of the ductus cochlearis.

The ligamentum spirale was poor in cells and showed some vacuolization. Beneath the beginning of the stria vascularis there was a mounded bowing into the lumen of the ductus cochlearis which consisted mainly of connective tissue. Corti's organ of the second and third coils was in good condition. The most marked change was in the spiral ganglion of the basal coil. This consisted mostly in the great paucity of ganglion cells. The cells still present were shrunken, with little protoplasm and small or no nuclei. In the nerve canals there was a marked thickening of the inner periosteum which was due to connective tissue with hyaline degeneration. The nerve bundles lying in this hyaline sheath were atrophic. In many canals they were entirely absent. The canal was then filled with the previously mentioned hyaline connective tissue mass. This hyaline connective tissue sheath followed the nerves into the lamina spiralis ossea.

The nervus cochlearis was well retained, but where it entered the tractus foraminulentus it rapidly diminished in thickness.

The observations in the left temporal bone were identical with those in the right.

Thus, in this case, there is the picture of a man who sustained injuries to the head twelve and twenty-two years before his death from an intercurrent infection. His hearing had gradually diminished during these years, and at section marked atrophic degenerative changes were found in the neuro-epithelium of Corti's organ and of the spiral ganglion, particularly in the vicinity of the basal coil of the cochlea. The end arborization of the cochlear nerve was degenerated and attenuated, and the fine bone canals housing these nerves were partly obliterated by a hyaline connective tissue. The general observations were, therefore, similar to those found by Alexander forty-four days after the accident, only more extensive, and, as Theodore points out, they were similar to

the changes which Manasse found in all his cases of progressive labyrinthine deafness.

Equally illuminating along this line is the report of Manasse.[24]

CASE 4.—A man, aged 39, had been injured fifteen years before death by a fall on the head; complete deafness had resulted. Death was caused by pneumonia. The observations were as follows:

1. Nothing pathologic was found in the skull or dura.

2. Fine fissures were seen microscopically but not macroscopically in both petrous bones in the labyrinth region. These affected on both sides the external wall of the labyrinth running through the round and oval windows. They were not united by bone or callous but were filled with connective tissue. The cochlea was spared by the fissures.

3. A bony new growth was seen inside the labyrinth which was symmetrical on both sides. This process was the same in the cochlea as in the vestibule, although the vestibule was hit harder than the cochlea, which fact can be referred to the localization of the fissures. The membranous semicircular canals were entirely obliterated by the new growth. Manasse called this process a periostitis ossificans.

4. The fourth significant observation was the atrophic degeneration of the nervous elements, the ductus cochlearis being most affected. There was a high grade degeneration of Corti's organ and also of the cochlear nerve and its branches. Similar degenerative changes were seen in the ganglion spirale.

One significant feature in this examination was that fine fissures often existed which could not be made out macroscopically, a point which Schönbauer and Brunner also stressed. A second significant observation was that after fifteen years the fissures were not united by bony callous; this fact will assume particular importance when I comment on its relation to the prognosis of these cases.

So all the pathologic changes in human beings are seen to be a regular sequence of events, consisting of hemorrhages in various places, the site of predilection being the perilymph spaces in the basal coil of the cochlea, in the vicinity of the round window. The exudation or transudation of blood is probably partly absorbed and partly organized, leading to atrophy of the finer nerve branches and degeneration of the neuroepithelium of both cochlea and vestibule, and always most marked in the region of the basal coil of the cochlea. The significant fact in the reports of cases in human beings is that the pathologic changes are almost always symmetrical and affect both temporal bones.

In discussing Barnick's cases in which the bony labyrinth was intact and nothing pathologic was found in the endolymph, Passow [1] concluded that the changes in the labyrinth were caused by concussion or commotio and not by fracture. While it is true, as Manasse and Schönbauer and Brunner pointed out, that fine fissures are often missed unless a careful microscopic search is made for them, nevertheless, one must find the cause for many of the hemorrhages in the labyrinth in a concussion or commotio caused by the violent movement of the entire head but more

particularly by the pressure of the brain against the saccus endolymphaticus. This is all the more reasonable because, as has been pointed out, the weakest portion of the entire fluid system of the inner ear is the region of the basal coil and the vestibular portion of the cochlea, in the vicinity of the round window, and it is this area in which the hemorrhages find their greatest expression and which subsequently suffers the most by reason of atrophic degenerative changes.

EXPERIMENTAL PATHOLOGY.

As Brunner [8] rightly concluded that the pathology of head injuries in cases in which the patients recover is probably different in many ways from that in cases of severe trauma in which the patients succumb, an attempt must be made to gain by animal experimentation some knowledge of what has taken place in the skull and ears of those injured persons who recover. Work along this line has been done by Stenger,[11] Jakob,[25] Brunner,[8] Uffenorde [26] and Yoshii.[27]

Stenger attempted to find out if, after a mild injury, there were changes in the inner ear analogous to those in concussion of the brain. He experimented with rats by striking their heads with light, heavier and still heavier blows, attempting, however, to avoid fractures of the skull or of the temporal bones. The animals became partially or completely unconscious, from which state they recovered after a time. The animals were then killed by careful bleeding, some as late as from three to four weeks after the injury. The microscopic examination showed hemorrhages into the inner ear in all twelve rats, depending, in degree, on the force of the blow used.

In the more mildly injured rats there were (1) hemorrhages in the region of the round window, and (2) hemorrhages in the basal coil of the cochlea, while the vestibule and semicircular canals remained free.

In the somewhat more severe injuries there were (1) more marked hemorrhages into the cochlea extending up to its very tip, but mainly in the scala tympani; (2) more marked hemorrhage at the round window; (3) mild hemorrhage inside the ductus cochlearis, and (4) occasional hemorrhages into the ampullae.

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25. Jakob, A.: Experimentelle Untersuchungen ueber die traumatische Schädigung des Zentralnervensystems, in Nissl-Alzheimer: Histologische und Histopathologische Arbeiten über die Grosshirnrinde, 1913, vol. 5, p. 182; Zur Klinik und Pathogenese der Postkommotionellen Hirnschwäche, München. med. Wchnschr. **65**:932, 1918.

26. Uffenorde, W.: Ohrenbefunde bei tierexperimentelgesetzten Schädelschussverletzungen, Ztschr. f. Laryng. Rhin. **15**:139, 1927.

27. Yoshii, U.: Experimental Study of Traumatic Injuries of the Ear, Zentralbl. f. Ohrenh. **9**:561, 1910-1911.

In the most severe injuries the hemorrhages seen were the same as in the second group, but, in addition, in some cases, the round window appeared torn and there was marked bleeding between the fibers of the acoustic nerve itself. Not in any of the animals was the temporal bone itself injured.

In an endeavor to find out what changes take place in the inner ear in commotio cerebri et auris internae, Brunner [8] repeated the original experiments of Stenger, using guinea-pigs, however. In these animals he could not find any definite changes in the neuro-epithelium of the inner ear, but he did find pathologic changes in the blood vessels, in the perilymph and endolymph spaces, in the aqueducts and in the membrane of the round window.

1. There was marked engorgement of the blood vessels in the basal portion of the cochlea. In the perivascular spaces, there was an exudate which stained with eosin and an increase in the number of lymphocytes. These vascular changes were seen in all the animals, though not always to the same degree.

2. Petechiae were noted between the nerve bundles of the maculae and cristae. These were not abundant. In the scalae, however, petechiae were more extensive. Brunner did not regard these petechiae as suffocation processes, as did Alexander, because Brunner's animals were killed in quiet narcosis by bleeding the heart. He thought that these petechiae were due to a diapedesis, because cut vessels were not seen.

3. The aqueductus vestibuli rarely showed any changes, though in one case its walls were edematous. The aqueductus cochleae, however, was filled with blood and exudate in all cases except one. The blood and exudate must have arisen from the perilymphatic spaces of the inner ear.

4. Hemorrhages were seen at the origin of the membrane of the round window. These were regarded by Brunner as due to a wave motion of the perilymph and endolymph at the time of the impact, because, just at this moment, the intracranial pressure is increased. He regards this excursion of the round window membrane as an ectasis of the cochlear aqueduct which serves as a protective mechanism for the more delicate neuro-epithelium.

Uffenorde's [26] experiments are not quite as important in connection with the problem, because he produced his injuries of the ear in rabbits and dogs by shooting them through the skull, although not directly through the region of the ear. He found the following changes in the inner ear:

1. Blood was almost always found in the aqueductus cochleae.

2. There were not any tears in the round window.

3. The membranous portions of the cochlea and vestibule were badly torn and disfigured in the more severe injuries.

4. Blood was found in all the perilymph and endolymph spaces, particularly in the macula region, and in the ampulla of the posterior semicircular canal.

5. The walls of the cochlear and vestibular labyrinth showed many fractures.

6. Hemorrhages into the internal meatus were always noted. These were mainly subdural, subarachnoidal and located between the nerve fibers. They affected all three nerves but mainly the facial nerve.

Jakob [25] experimented with twenty-four rabbits and three monkeys. One or more blows were struck with a hammer in the left parietal region in these animals. The force of the blows was graduated so that the different degrees of injury could be studied. The animals were kept alive for varying periods, and then histologic examinations were made of the brain and spinal cord. Every effort was made to prevent gross lesions so that in only one of the animals was the skull fractured, and in only three were there any meningeal hemorrhages. When these hemorrhages did occur, they affected the medulla.

As to the traumatic injuries of the brain substance itself, hemorrhages and softening were found in several animals.

1. The favorite site of punctate hemorrhage in trauma of the brain was the medulla and upper cervical cord.
2. The looser and more vascular gray matter showed more tendency to hemorrhage than the white mass of fibers.
3. Microscopic foci of softening were present between the gray and white matter of the corpus striatum.
4. Foci of softening were observed around the larger and smaller blood vessels.
5. Foci of softening were noted in the floor of the fourth ventricle and near the central canal.

Jakob thought that the sudden movement of the cerebrospinal fluid was of importance in causing the original lesions of which the softenings were the later sequelae. His experimental evidence showed that there was not any direct relation between the functional or anatomic disturbances and the severity of the trauma.

Brunner [8] attempted to find out how the central areas of the cochlear and vestibular nerves react to skull trauma, by subjecting guinea-pigs to light injuries of the head which did not even elicit the symptoms of concussion of the brain. It was to be expected that in the nuclei of the nervus cochlearis and the nervus vestibularis, which lie superficially at the surface of the medulla, the results of the trauma could be found. The animals were killed at various periods after the injury.

By the use of Nissl's method of staining, Brunner found degenerative changes which took place mainly in the small cells of the nucleus trianularis, in the nucleus of von Bechterew, in the tuberculum acusticum and in the posterior corpora quadrigemina, while the larger elements of Deiters' nucleus suffered only if the animals lived a long time after the trauma. Brunner believed that in human beings frequent degenerative changes must take place in the nuclei of the cochlear and vestibular nerves after injuries of the head.

Drawing on the work of Ricker,[14] Brunner showed that the effects of the injury on the vascular system of the blood depend on changes in the irritability of the vasodilators and the vasoconstrictors, and that this changed irritability produces localized areas of altered circulation.

The effects of the mechanical irritation of the vasomotors in the order of their severity are: (1) a dilatation of the blood channel with increased rapidity of flow or fluxion; (2) a narrowing of the blood stream or ischemia; (3) first grade of dilatation of the vessels and slowing of the stream or an inflammatory condition; (4) second grade of dilatation of the vessels with slowing of the stream, a condition of prestasis and stasis.

In the stage of stasis and prestasis exudation of blood cells and blood serum occurs.

Knauer and Enderlen,[15] attempting experimentally to produce a condition of commotio cerebri in dogs by striking them on the head with a heavy hammer, also came to the conclusion that after the trauma a state of traumatic paralysis of the vasoconstrictor nerves arises, which they think is due to damage of the midbrain. They found in their experimentally produced commotio cerebri that there were vascular changes in the circulation characterized by (1) a reduction in the irritability of the vasomotors; (2) the spontaneous appearance of stasis and petechiae, and (3) the occasional exudation of cell-free fluid.

If the vasomotor changes operate for a long time, they must produce changes in the cellular tissue supplied by the affected vessels, the end-result of which is necrosis and scar formation.

Brunner believed with Jakob that some of the degenerative changes after injury are due to direct insult to the nerve tissue by the trauma itself but that the larger part of the damage is due to the effect on the nerve tissue of the injury to the vasomotor center.

As the vessels of the inner ear are in direct communication with those of the brain and are similarly innervated, Brunner thought that a condition of prestasis could exist in these vessels even as it exists in the brain in commotio cerebri. In his experimental animals he found an edema in the perivascular connective tissue of the modiolus. This condition was, however, never found in the connective tissue of the cristae or maculae. Here the exudate was poured out into the hollow spaces.

Brunner designated these vascular changes in the inner ear by the term "otitis interna vaso-motoria" with the understanding that the word "otitis" does not have any connection with infection but is, in this instance, a disturbance in the circulation of the ear due to an involvement of the vasomotor center.

It is seen, thus, in all this experimental work in which an effort was made to simulate, as much as possible, injuries of the head sustained by human beings, and in which the animals could be killed at any period after the injury, that the changes, in the main, were similar to those described under the section on pathologic changes in human beings. The changes in the inner ear were always more marked in the cochlea than in the vestibule and consisted, for the most part, of hemorrhages.

These were always most marked in the basal coil of the cochlea and in the vicinity of the round window.

Furthermore, these experiments have shown, as Brunner pointed out, that in these cases of injuries to the head one cannot regard the ear as being injured in an isolated fashion. It must be remembered that in every instance the brain itself is also injured. The stability of the vasomotor control is lost. The nuclear territory at the floor of the fourth ventricle suffers particular damage, whether it be from direct insult to the nerve tissue or only from degenerative changes in the vestibular and cochlear nuclei caused by the vasomotor changes in the circulation of the blood in this territory, or from both of these causes.

The experiments also show that there is little relation between the severity of the injury and the degree of the damage. The animals were, for the most part, subjected to light injuries, and in only a few instances were the bones of the skull or ear fractured.

CLASSIFICATION OF INJURIES OF THE EAR

In the classification of the injuries of the ear which follow trauma to the head, a rather difficult problem presents itself. One is obliged to classify the injuries in the interior of the ear and brain, in patients who recover from their accidents, without always being able to know just what damage has been done. One can, however, avail oneself of the pathologic observations in the patients who die and in those few patients who have survived their injury of the head for a long time. Of particular help will be the study of the experimental lesions produced in animals.

Passow [1] divided the pathologic changes of ear injuries into: (1) fractures of the labyrinth capsule, and (2) hemorrhages into the labyrinth or commotio labyrinthi.

Stenger [11] classifies his cases as follows: (1) damage to the inner ear with injury to the bony labyrinthine capsule; (2) damage to the inner ear without any damage to the bony labyrinthine capsule; (3) damage to the inner ear without any demonstrable lesion of the skull; the first group comprise the transverse fractures of the pyramid. The second variety is caused by longitudinal fractures of the temporal bone. The third group of cases Stenger calls "Labyrintherschütterung."

Mauthner [28] divides his cases according to Manasse's classification into:

A. Cases in which recovery is functionally complete or almost so. These are subdivided into:

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28. Mauthner, O.: Die traumatische Erkrankungen des inneren Ohres, Arch. f. Ohrenh. **87**:146, 1911.

(*a*) Mild cases in which hearing is normal or almost normal, but with definitely demonstrable labyrinthine disturbances caused by a commotio labyrinthini.

(*b*) Severe cases of typical basal skull fractures in which, nevertheless, there is a return to a normal condition (rare).

B. Cases in which there is severe lasting functional damage; traumatic deafness, complete or incomplete.

(*a*) As the result of petrous bone fracture.

(*b*) As the result of a commotio labyrinthi. (These forms can only be divided anatomically, not clinically.)

To all three of these classifications the objection can be raised which Brunner made regarding Stenger's division: that they do not take into consideration the concomitant injury to the brain which is always present in these cases in addition to the injury of the ear. These cases cannot be regarded as isolated injuries of the structures inside the temporal bone. Brunner, therefore, classifies as follows: (1) commotio cerebri; (2) commotio cerebri et auris internae; (3) fracturae ossis petrosae.

In group 1 will be found the cases in which the clinical observations in the ear depend on changes in the central auditory area (the central vestibular system). In these cases there would not be any loss of hearing.

In group 2 are the cases which, in addition to the symptoms arising from the nuclei, also present symptoms arising from damage to the membranous canal linings. These cases would always present some loss of hearing in addition to the vestibular symptoms exhibited by the cases in group 1.

Group 3 includes all fractures of the temporal bone.

This classification is probably more comprehensive than any yet offered, but even with this classification great difficulty will be encountered clinically in making cases fit the different groups. It will be found particularly hard in many instances to say whether a patient examined clinically belongs in group 2 or whether the case is one of longitudinal fracture of the temporal bone.

SYMPTOMS AND SIGNS

I have now to present the results of the clinical examination in forty-two cases of injuries of the head collected over a period of a year and a half. Of this number thirteen were examined during the first ten days following the injury. Seven more were examined during the first month, and the remaining twenty-two were first seen at periods varying from one and one-half months to three years after the original injury. Some of the patients were examined repeatedly, but many were seen only once. The injuries were the result of a variety of different types of accident, as I have outlined earlier.

Some were severe injuries, and others were apparently minor accidents. Eight of the forty-two patients did not lose consciousness, and in five loss of consciousness was not noted.

The severity of the impact does not seem to have had much bearing on the production of otologic symptoms in my series of cases. In the eight cases without concussion, there was one (case 5) in which there was bleeding from one ear and from the mouth. The patient later developed a bilateral reduction of hearing for the C 5 (4,096 double vibrations) tone and a hypo-irritable labyrinth on both sides, more marked on the left, the side on which his ear bled. A report of his case follows:

CASE 5.—*History.*—Marcellino Burrera, aged 23, while working next to a freight car on April 22, 1927, was struck in the region of the right ear by a passing wagon and the left side of his head was slapped up against the freight car. He was not unconscious. There was bleeding from the left ear and possibly also from the right, although a note from his attending physician states that the accumulation of blood in the right external canal was probably due to a wound of the anterior portion of the right helix. There was also a little bleeding from the nose. He was taken to the hospital where he remained in bed for four weeks. He did not have any actual headache while in the hospital but complained of his head feeling heavy. During the first two weeks of his stay in the hospital he was dizzy.

On May 19, 1927, he was only slightly dizzy; the vertigo was likely to come on in spells when he got out of bed in the morning or lay down at night. He did not have a headache, but complained of slight difficulty of hearing in his left ear.

Examination.—Spontaneous Symptoms: The gait with the eyes closed was normal. The Romberg sign was negative. Spontaneous nystagmus was not present in any direction, but this could be produced by shaking the head, after which a rotary-horizontal nystagmus appeared to the left in the end position. Spontaneous past pointing and deviation of the right arm was not observed, but positive spontaneous past pointing and spontaneous deviation of the left arm to the left were noted.

Eyes: The pupils were equal and reacted to light and in accommodation. Paralysis of the ocular muscles was not observed.

Face: Facial paralysis was not noted.

Right Ear: There was a stellate scar at the upper front end of the helix. The external canal was clear, and the tympanic membrane showed some retraction.

Left Ear: The external canal was clear. Some old blood clots were present in the depth of the canal. There was some redness of Shrapnell's membrane. A healed scar was seen leading from the umbus downward and backward in a semicircle to the margo.

Tests of the hearing yielded the following results:

Tests of Hearing	Right Ear	Left Ear
C (32 double vibrations)...	Hearing	Hearing
C (4096 double vibrations)	Hearing	Hearing
Whisper	At 20 feet plus	At 20 feet plus
Watch	At 20 inches	At 25 inches
Weber.....		To left
Rinne	Positive 12; 28	Positive 12; 31
Schwabach	Six seconds short	Normal

In the audiogram (chart 2), the absolute similarity of the curve in the two ears and the fact that the hearing is more damaged in the right ear, which did not bleed, than in the left should be noted.

Vestibular Tests.—Right Ear: Fifteen cubic centimeters of water at 15 C. produced in twenty seconds a slight horizontal-rotary nystagmus to the left lasting seventy seconds. Deviation was not observed in either arm, and only slight vertigo was noted.

Left Ear: Fifteen cubic centimeters of water at 15 C. produced in fifty-five seconds only the slightest horizontal-rotary nystagmus to the right, consisting of a few twitches. Deviation of either arm and vertigo were not noted. Both labyrinths were hypo-irritable, the left more than the right.

In this case it is seen that, while the patient did not present a severe type of injury when reckoned from the point of view of loss of con-

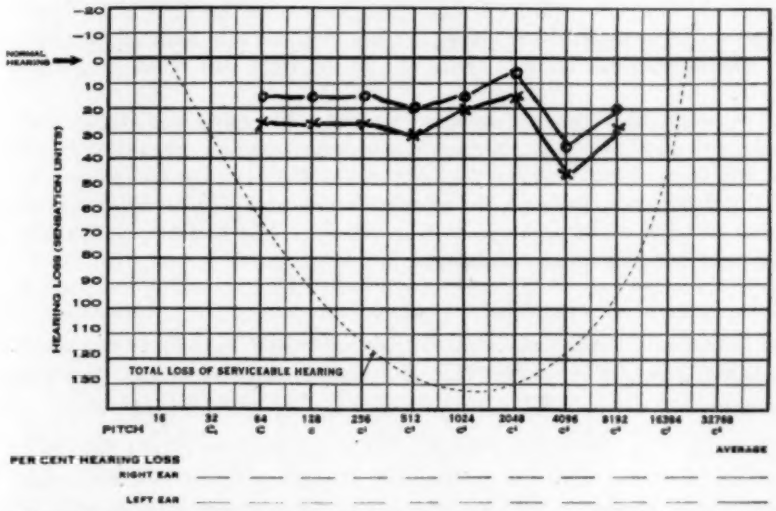

Chart 2 (case 5).—Audiograms of both ears. In this and the following charts, the circle indicates the left ear, and the x indicates the right ear.

sciousness, he did, however, have bleeding from one ear, a permanent loss of hearing in both ears, especially for the tone of 4,096 double vibrations and other tones in this range, and an inequality in the irritability of the two labyrinths, one of which was decidedly hypo-irritable.

Other men have noted this discrepancy between the severity of the trauma and the occurrence or nonoccurrence of hearing and vestibular symptoms. Moriez [29] did not find any connection between the severity of the trauma and the occurrence or failure of vertigo. In Mygind's [2] series of 142 cases, concussion occurred in only a little more than 50

29. Moriez, A.: Le vertige, sequelle frequente des traumatismes de la tête; Petit étude statistique, Rev. d'oto-neuro-ocul. **4**:406, 1926.

per cent. It did not have any connection with the later development of cochlear or vestibular symptoms. In fact, some of his injuries were extremely slight, the patients entering the hospital for other purposes. Passow [1] called attention to the fact that nerve deafness is common after all basal skull fractures, and that a relation cannot always be established between the degree of this deafness and the severity of the injury. In this connection, the following case reported by Urbantschitsch [30] is interesting.

Case 6.—*History.*—A man was struck down by a blow in the right parietal region. He was not unconscious, and no symptoms developed except severe pain of several days' duration at the site of the injury. He returned to work. Seventeen days thereafter, while at work, he experienced sudden severe tinnitus and deafness in the left ear, together with vertigo and vomiting.

Examination.—The voice was heard at 6 meters on the right side; there was complete deafness on the left side. The left vestibule was absolutely nonirritable to caloric irritation. Marked nystagmus to the right was noted in positions I and II.

Two weeks later, little vertigo was observed. The nystagmus to the right was obtained in position I only. A trace of nystagmus appeared after the administration of 3 liters of cold water in the left ear, and slight hearing was present.

Six weeks after the onset, the caloric reaction was positive with the administration of 1.5 liters of cold water. The voice could be heard up to 6 meters by each ear. The patient was discharged.

Urbantschitsch thought that these symptoms were caused by sudden hemorrhage into the left inner ear seventeen days after the accident, probably owing to the fact that the vessel, injured at the time of the accident, did not actually rupture until seventeen days later, when a rise may have occurred in the patient's blood pressure while he was at work. Another interesting point is that the trauma affected the ear opposite the point of impact. The patient apparently was not injured sufficiently to be taken away from work.

In my own series of forty-two cases bleeding occurred from one or both ears in nine cases. In twenty-one cases bleeding did not occur from either ear, and in twelve cases the question of bleeding from the ears was not noted in the history. In a series of 408 cases of fracture of the skull, Borden [12] found hemorrhage from one or both ears in 221 cases, and according to Graf, cited by Passow,[1] bleeding from the ear occurred in seventy-seven of ninety cases of basal skull fracture. Phelps,[13] in his series of 1,000 cases of fracture of the skull, found this symptom present in 285 cases, or 28.5 per cent. Of these, 166 recovered, and 119 died. Phelps held that hemorrhage from an ear is an absolutely diagnostic sign of a fracture of the temporal bone.

30. Urbantschitsch, E.: Labyrinthblutung 17 Tagen nach einem Schädeltrauma, Oesterreichische otol. Gesellsch., January, 1923.

Kobrak [3] and Passow,[1] while they agreed in this opinion, did not regard hemorrhage from an ear as always diagnostic of a basal skull fracture. They maintained that the temporal bone may be fractured without any basal skull fracture, and Kobrak even went so far as to say that the bleeding may occur from a ruptured tympanic membrane, which belief most authors deny. To my mind, this sign offers indubitable evidence of a fracture of the temporal bone. However, as Ulrich [13] pointed out, this fracture of the petrous temporal bone must be of the longitudinal type which projects itself through the middle ear, through the tegmen antri or tympani and then through the margo tympani to end in the external canal wall. A transverse fracture of the pyramid involves the middle ear only rarely when it affects the promontory wall, so that in this type of fracture, one will not expect to see any hemorrhage from the ear. That a patient may have a serious injury to the head, with bleeding from both ears, and escape with little ultimate damage to the ear is illustrated by the following case:

Case 7.—*History.*—Charles McCormick, aged 12, struck by an automobile on April 11, 1926, was unconscious and bled from both ears. He was taken to the Marquette General Hospital, where he remained for four weeks under the care of Dr. J. A. Purtell. A diagnosis of fracture of the skull and fracture of the clavicle was confirmed by the x-ray.

According to his mother, when he was brought for examination on July 12, he complained only of general nervousness and frequent headaches. Vertigo was not noted.

Examination.—Eyes: The vision was 20/20 in each eye. The pupils were small, round and equal. They reacted to light, both direct and consensual, and to accommodation. The action of the extra-ocular muscles was normal. Convergence was good. The media were clear. The fundi were negative, except for a slight engorgement of the veins and a watered silk appearance of each retina.

Ears: Both external canals were filled with cerumen, which was removed. Both drums were negative.

Tests of the hearing gave the following results:

Tests of Hearing	Right Ear	Left Ear
Watch	At 45 inches	At 45 inches
C (32 double vibrations)	Hearing	Hearing
Galton whistle	To O	To O
Rinne	Positive 10; 24	Positive 7; 31

The audiogram for this case is shown in chart 3.

Vestibular Tests.—Spontaneous nystagmus was not present. Spontaneous past pointing was not observed. The Romberg reaction was negative. The gait with the eyes closed was normal.

Brunner [8] also noted in ten of his patients who had bleeding from the ear that cochlear and vestibular symptoms were not present.

The most common complaint offered by the patient at the time of the otologic examination is headache. This symptom is usually not

constantly present, varies a great deal in its character and comes on in attacks. Rapid change in position, particularly of the head, undue exertion, exposure to the sun and indulgence in alcohol are factors which tend to bring on the attacks of headache. They are also often associated with vertigo.

Deafness was complained of by fourteen patients, tinnitus by eight and double vision by three. The diplopia was transitory except in one case, that of a man who was struck in the right frontal region by a heavy timber while he was at work in a mine. At the time of examination, he had a loss of vision of the left eye to 20/200, with a well developed optic atrophy, limitation of the left external rectus and left superior oblique muscles, facial paralysis on the left side and a reduction of

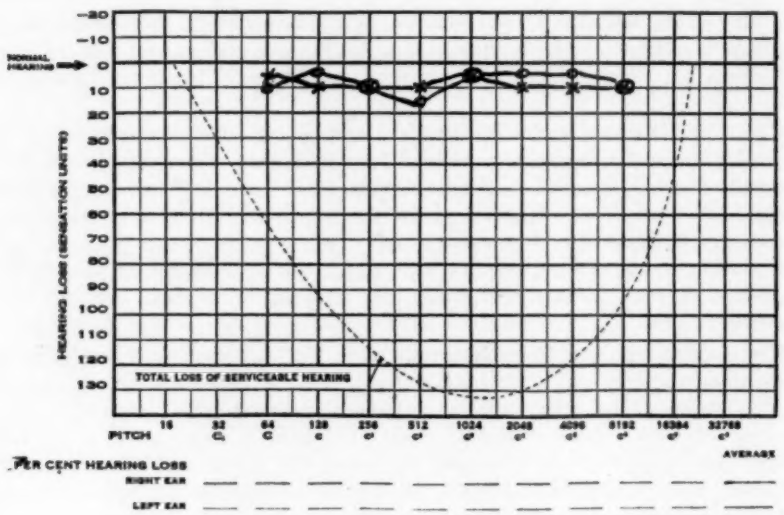

Chart 3 (case 7).—Audiograms of both ears.

hearing in the left ear over the entire tone range, but especially marked in the upper tonal range, C 5 being cut down 80 sensation units on the audiogram and C 6 not being heard at all.

I come now to vertigo, a symptom often complained of by patients with injuries of the head, and one which is of great importance in making a diagnosis of the condition from an otologic standpoint. Great care must be exercised in eliciting a history of vertigo from the patient. He should not be asked any leading questions about this symptom. If the history is patiently taken, the patient will usually volunteer the information about this complaint. He must then be asked to describe carefully what he means by being dizzy, in order to bring out, if possible, a rotary character of the vertigo, but great care must be exercised not to suggest to the patient in any way that his vertigo should have a rotary character.

It is important to establish the fact that the vertigo has a rotary character, because this fixes it beyond doubt as of labyrinthine origin. The patient will tell you that external objects move about him or that he seems to turn or that his head keeps going around. According to Rhese,[31] there is usually a movement of the external objects toward the slow component of the existing spontaneous nystagmus. When the external objects rotate toward the sound side, Rhese, together with Schede, believed that a cerebellar lesion is indicated, ceteris paribus.

Brunner [8] contended that the vertigo need not be of the rotary type to establish its vestibular character if it comes on in attacks accompanied by typical spontaneous vestibular nystagmus.

When the vertigo is due to severe hemorrhage into the labyrinth (Bárány [32]), it begins immediately, is usually continuous and is severe, with all the characteristics of vertigo after sudden exclusion of the labyrinth, i. e., intense nystagmus to the normal side, falling to the diseased side, increase of the vertigo and nystagmus from every movement. The severe vertigo usually diminishes in from eight to fourteen days, and finally disappears entirely, if the labyrinth of the other side has not been damaged. Such continuous dizziness was present in two of my patients during the first week or two following their injuries. One was a patient (case 1), who sustained a transverse fracture of the pyramid and whose case has been reported in detail. The other was a young lady who sustained a sęvere trauma of the head in an automobile accident, and who subsequently developed a marked loss of hearing in her left ear.

If the vestibular system has not been greatly damaged or destroyed, the vertigo is not continuous but comes on in attacks. This type of vertigo is usually not noticed by the patient until he gets out of bed for the first time or until he leaves the hospital. Passow [1] called attention to the fact that it might appear quite late, and be first noticed when the patients go to work, when they are above the ground level, when they bend over or when they work energetically. Passow is also the author of the statement that the attacks may last for years after the injury.

Bárány [32] noted two kinds of attacks of vertigo.

1. The first variety appears without any visible cause. It is generally severe and lasts from one-half hour to several hours. Any movement increases the vertigo. During the attack, there is usually intense nystagmus to the affected side, with falling to the normal side.

31. Rhese: Ueber die traumatische Läsion der Vestibularisbahn, insbesondere über den Sitz der Läsion, Ztschr. f. Ohrenh. **70**:262, 1914.

32. Bárány, R.: Untersuchungen über das Verhalten des Vestibularapparates bei Kopftraumen und ihre praktische Bedeutung, Verhandl. d. deutsch. otol. Gesellsch. **16**:252, 1907.

2. The second type of attack is milder than the first, lasts only a short time and is caused by getting out of bed, bending over, lying down, rapidly turning the head or the entire body, bending the head to one side, shaking the head, undue exertion, sudden changes of temperature, exposure to sunlight, indulgence in alcohol and tobacco and slight digestive disturbances. Most of the vertigo of my patients was of this type. The vestibular apparatus of these patients must be in a highly sensitive and irritable condition, and the variety of causes which can bring on an attack would rather point to a vasomotor disturbance in the vestibular system as the causative factor.

In those cases of vertigo which develop immediately after injury, in which deafness and tinnitus do not exist, it will be hard to distinguish between an injury to the vestibular portion of the labyrinth only and an intracranial injury of the nuclear region of the vestibular nerve.

Bárány found that the vertigo of the neurotic patient is often accompanied by severe and long continued nausea and vomiting, mental excitement and anxiety, stupor and approximate loss of consciousness. Brunner [8] stated that in cases of commotio cerebri with symptoms in the ears, the vertigo complained of is of vestibular origin even in the absence of a spontaneous nystagmus, if it is accompanied by a caloric hyperirritability, and if a nystagmus appears after rapid movements of the head. The assumption of some authors that a vertigo must be associated with a spontaneous nystagmus to be called labyrinthine goes too far, in his opinion, because many of the patients do not have a constantly present spontaneous nystagmus. The nystagmus may be present on one examination and absent on another. Mygind [2] made a statement which I can heartily endorse, when he said that the vertigo is only exceptionally of severe nature and is always accompanied by headache.

One of my patients who had sustained a slight injury, when examined two months after his injury complained of constant dizziness. Constant vertigo, appearing this late, is, I think, a neurotic symptom, in spite of the fact that this particular patient had a marked spontaneous nystagmus, a hyperirritable labyrinth on the right side and a slight bilateral loss of hearing in the range of a tone of 4,096 double vibrations (*C* 6), for the complaint of constant vertigo, as also of constant headache, after the first two or three week period should lead one to suspect a neurosis, either isolated or added to the labyrinthine lesion.

Vertigo, then, is one of the cardinal symptoms of involvement of the vestibular system. Its labyrinthine character is established by the fact that it is rotary, or if not rotary by the fact that it is accompanied by nystagmus, either spontaneous or induced by the head movements test, or, according to Brunner, if it is accompanied by a hyperirritability of the labyrinth.

Another spontaneous symptom of great importance is spontaneous nystagmus. If the vestibule has been destroyed or greatly damaged, a strong spontaneous nystagmus toward the sound side, associated with marked vertigo, occurs immediately after the injury. This symptom appears in any position of the eyes but is more marked when the patient is looking toward the sound side, and it is aggravated by any movement of the head. It is also associated with marked vertigo and nausea. In this class of cases the nystagmus, together with associated vertigo, will subside after the lapse of one or two weeks.

In other injuries of the head, a spontaneous nystagmus to the sound side is also found shortly after the accident. This condition, however, disappears by the time the patient has recovered consciousness, as Bárány,[32] Kobrak [3] and Rhese [31] have noted. The nystagmus which is elicited at a later date is usually unilateral; if it is bilateral, it is usually stronger toward one side, or if it is bilateral and of equal intensity to both sides, it will become more marked to one side after the so-called head movements test. In this test, the patient's head is grasped between the two hands of the examiner; then, after two or three gentle forward and backward movements, to determine whether it is relaxed or not, the head is suddenly and sharply bent backward, and the patient is immediately directed to look to one side and then to the other. The production of, or change in, the spontaneous nystagmus by this maneuver was given great value by Bárány. Brunner, on the other hand, did not find it of any great help. The true vestibular spontaneous nystagmus is always horizontal and rotary. A bilateral spontaneous nystagmus is occasionally seen in normal persons, but this is usually a pure horizontal type, is always equal to the two sides, is not associated with vertigo and is never influenced by the head movements test.

Brunner [8] studied sixty patients with commotio cerebri of whom thirty-four showed symptoms relative to the eighth nerve. Of these, all but eight exhibited spontaneous nystagmus, and Brunner believed that some of the eight would have exhibited nystagmus if they had been examined more frequently, for the spontaneous nystagmus is not constant and, therefore, can be missed when only one examination is made. Rhese [31] found spontaneous nystagmus in all but one of his patients who were examined early, and in 85 per cent of the patients who were examined at a later date. Brunner was not able to formulate any rule regarding the direction of the nystagmus with respect to the injured ear. Mygind, on the other hand, believed that the nystagmus was directed toward the injured ear. Rhese believed that the spontaneous nystagmus, when present, was caused by a hypo-irritable condition of one labyrinth. In other words, the nystagmus was due to a decompensation between the two labyrinths. In this latter statement he was probably correct, but as Brunner found, and as I shall demonstrate in some of my cases, a

hyperirritable condition is often present. I am, however, firmly convinced that whether it is due to a hypo-irritability or to a hyperirritability of the labyrinth, the nystagmus is caused by a decompensation between the two labyrinths, by a decompensation between the two central vestibular nuclei or by both conditions.

In my own series, spontaneous nystagmus was not found in nine cases. It was present in twenty-nine cases. In five cases it was bilateral and equal on the two sides; in twenty-four cases it was unilateral, or if bilateral, then more marked to one side; in four cases it was not present on examination, but could be produced by the head movements test. In all the examinations, the tests were made in the lateral or end position of the eyes, position I.

In connection with the question of spontaneous nystagmus and of vestibular irritability the following case is of interest:

CASE 8.—*History.*—Joseph Gilbert, aged 39, was struck on the vertex by the band of a descending bucket, while at work in a tunnel on July 27, 1927, two weeks before I examined him. He was knocked down and was unconscious for one-half hour. He stated that his nose bled immediately after the accident, but that his ears did not. However, he did not stop work. He said that he had a headache immediately after the accident.

The headache was more or less continuous. He had dizzy spells two or three times a day, lasting two or three minutes each, during which everything in his head seemed to whirl. This made him uncertain on his feet, but did not cause him to fall.

Examination.—Spontaneous Phenomena: The Romberg sign was negative in both the regulation position and the heel to toe position. The forward gait with the eyes closed was slightly to the right while the backward gait was to the left. There was spontaneous past pointing of both arms to the left, and spontaneous deviation of the left arm to the left, but no deviation with the right arm. A spontaneous nystagmus to the right was obtained in the end position and also to the left in the end position, and the look to the left produced vertigo. The head movements test increased the nystagmus to the left and produced vertigo.

Ears: Both external canals were clear. The right drum was normal. The left drum was slightly retracted and showed a redness of Shrapnell's membrane, the posterior superior canal wall and the handle of the hammer.

Eyes: The pupils were equal and reacted to light. The media were clear. The fundi were negative. Ocular palsies were not observed.

Tests of the hearing yielded the following results:

Tests of Hearing	Right Ear	Left Ear
Whisper	At 30 feet	At 30 feet
Watch	At 30 inches	At 30 inches
C (32 double vibrations)..	Hearing	Hearing
C (4,096 double vibrations).	Normal	Two seconds short
Weber	Not referred	Not referred
Schwabach.....	Seven seconds short	Eight seconds short

The audiogram (chart 4) showed a reduction of the entire tone range, more marked in the left ear, and especially more marked in the range of the tone of 4,096 double vibrations.

Caloric Tests.—Right Ear: Five cubic centimeters of water at 15 C. produced in ten seconds a rotary-horizontal nystagmus to the left, lasting 135 seconds in positions I, II and III, and some slight vertigo.

Left Ear: Five cubic centimeters of water at 15 C. produced in ten seconds a rotary-horizontal nystagmus to the right lasting 150 seconds in positions I, II and III, and slight vertigo.

Both labyrinths were hyperirritable.

The interesting feature in case 8, as far as the spontaneous nystagmus is concerned, is that when the patient looked to the left the spontaneous nystagmus was accompanied by vertigo, and that after the head movements test the nystagmus was increased to the left and vertigo again appeared.

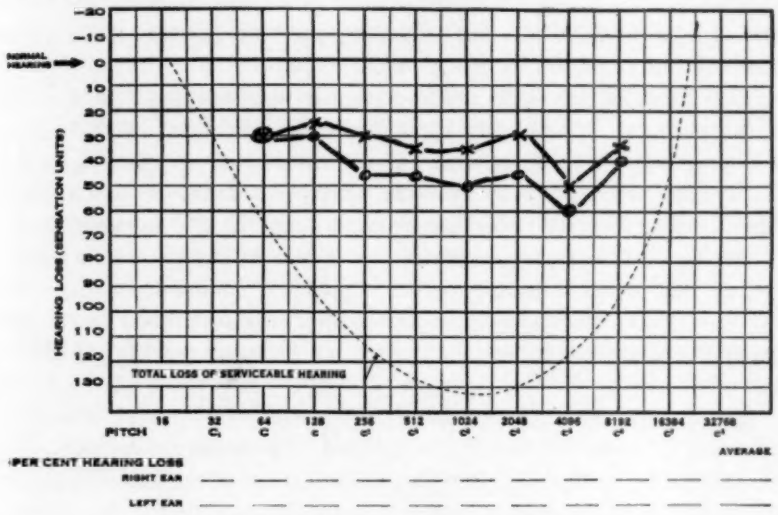

Chart 4 (case 8).—Audiograms of both ears.

In the study of the disturbances of equilibrium in my series of cases, nothing of great value in respect to the lesions of the ears was uncovered with the exception that twenty-eight of the forty-two patients exhibited some form of spontaneous past pointing or spontaneous deviation of the upper extremity. Tests for the pointing reaction were always performed in the upper extremity only and with the shoulders down. Bárány [33] recently brought out the deviation reaction (Abweichsreaktion) which consists in stretching both arms forward parallel and holding them there with eyes closed while the examiner marks their original position with his own arms and then pulls his arms back. If a spontaneous pointing reaction has been present then the arm or arms

33. Bárány, R.: I. Der Zeigeversuch; II. Die Abweichreaktion; III. Der Einstellungsversuch, Acta oto-laryng. **7**:139, 1925.

will slowly deviate in the direction which this past pointing has taken. Fischer and Wodak [34] held that this reaction was much more sensitive than the original pointing reaction, but pointed out that the normal person deviated outward slightly with both arms. They also made the statement, which I can corroborate, that at times the pointing reaction was negative, while the deviation reaction was positive, and vice versa.

Spontaneous past pointing or deviation to one side or the other was present in twenty-eight of my forty-two patients. In thirteen it was directed to the same side as the existing spontaneous nystagmus, and in seven it was directed to the opposite side. In six patients, the past pointing or deviation reaction was positive, while the nystagmus was found to be equal to the two sides, and in two patients, pointing deviations were present, but nystagmus could not be made out at the time of examination. In the remaining fourteen patients deviation and past pointing could not be made out.

The forward gait with the eyes closed was tested out in a number of the patients, because De Haan [35] pointed out that, as the legs are ordinarily used in walking and not in pointing at or grasping objects, the pointing reaction in them could better be tested out by having the patient walk forward with eyes closed than by the same method in which the test is carried out in the upper extremities. In fact, he showed that when a normal person receives a cold douche in one ear, and then walks forward, he deviates in his gait toward the side on which the douche was received, even as he past points and falls to that side.

In my series only eight patients presented any abnormalities of the gait, and in seven of these eight patients the deviation in gait was to the same side as the existing past pointing or deviation of the arms, but in one case the past pointing was to one side, while the deviation in gait was toward the other side.

Nor was the Romberg reaction found to have any particular value. This test was performed on twenty-eight patients, both in the standard position and with the heel of one foot to the side of the toe of the other, the position of Erb. The reaction was entirely negative in twenty-four cases, and in the four cases in which it was positive cerebellar involvement was strongly suggested in the traumatic lesion.

The reaction of the labyrinth to stimulation is of considerable importance in the diagnosis in the traumatic lesions of the ear. I have restricted my examinations to the use of the caloric test except in a few cases in which the turning tests were used because the caloric test

34. Fischer, M. H., and Wodak, E.: Principielle Betrachtungen zum Probleme des Zeigeversuchs, Acta oto-laryng. **10**:24, 1926.

35. De Haan, P.: On the Influence of the Semicircular Canals on the Gait of Man, Acta oto-laryng. **10**:63, 1926.

seemed contraindicated. This test, to my mind, offers much greater possibilities of testing each labyrinth separately than does the turning reaction. There are instances, however, such as case 1, when total exclusion of one labyrinth is suggested, in which the caloric test must be supplemented by the turning test, and also, at times, by the galvanic reaction. The caloric test is also more or less contraindicated by discharging ears. At the beginning of my investigation the mass irrigation of Bárány was used, but most of my patients have been examined by the Kobrak method.

In this method, only 5 cc. of water at approximately 15 C. is used. In normal persons this should produce a nystagmus to the opposite side, appearing in from fifteen to twenty-five seconds and lasting from sixty to 120 seconds, according to Brunner [8] and Wodak.[36] It will also almost always produce a past pointing, deviation and falling to the same side and has the decided advantage of causing little or no vertigo. Furthermore, as the same quantity of water at the same temperature is used in each ear, one can study not only normal irritability, hyperirritability and hypo-irritability, but also—which is important—differences in the irritability of the two sides, barring anatomic differences in the conducting apparatus of the two ears. If 5 cc. of the irrigating fluid does not produce any reaction, the quantity can be increased to 15 or 20 cc.; and if this amount is still insufficient, the mass irrigation of Bárány can be used. The resulting nystagmus is examined in positions I, II and III of the eyes, position III being toward the ear which received the douche, and position I being away from it. As my object is merely to determine the irritability of the vestibular system in its varying degrees, only the resulting nystagmus was taken into account, and I did not pay any particular attention to the induced pointing and falling reactions.

Alexander and Brunner [37] said that if any real anatomic disease exists in the vestibular nuclear region or in the conducting nerves, there can never be more than transitory hyperexcitability. Any anatomic disease in these areas would cause a decrease, and not an increase, of function. Diseases which only irritate the nerves or nuclei may cause hyperexcitability. Clinically, hyperexcitability occurs in cases of acute peripheral inflammations, in peripheral congestion, in central congestion and in central rise of pressure after trauma. In all these conditions the etiologic factor of the hyperexcitability appears to be the same, namely, a rise of pressure in the peripheral or central region, with the nerves and nuclei themselves anatomically intact. This irritation may also

36. Wodak, E.: Zur exakteren klinischen Differenzierung der Erregbarkeitsverhältnisse des Vestibularapparates, Acta oto-laryng. **9**:245, 1926.

37. Alexander, G., and Brunner, H.: Ueber Labyrinthäre Uebererregbarkeit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **3**:243, 1922.

involve the tracts which connect the nuclei of the eighth nerve with the nuclei of the eye muscle.

Hyperexcitability is caused by two factors which differ in localization. 1. Hyperirritability is, in the narrower sense, caused by pathologic changes in the inner ear and by pathologic changes in the peripheral sensory nerves up to the point of entrance into the brain stem. 2. Hypersensitiveness is caused by an abnormal functional condition of the central vestibular system.

Hyperexcitability may be caused by any pathologic change in the middle ear or labyrinthine capsule which causes abnormal conductivity in the nerves, either mechanically or by a rise of temperature from local inflammation, by acute or subacute inflammatory changes in the sensory terminals and, possibly, by changes in the motor conditions for motor reaction capacity of the cupolae and statolithic membranes. Changes in the endolabyrinthine pressure do not cause hyperexcitability, according to these authors.

For a diagnosis of either hyperirritability or hypersensitivity, Alexander and Brunner held that a history of characteristic labyrinthine vertigo is absolutely necessary.

Differences in labyrinthine irritability can easily be determined by Kobrak's caloric method which measures both the latent period and the duration of the nystagmus. The latent period is dependent on conditions of irritability of the peripheral end-organ or of the peripheral nerves. The duration of the nystagmus is dependent on the sensitiveness of the central nervous system. Hyperexcitability of the peripheral end-organ is usually associated with hypersensitiveness of the central nervous system and the same is true for the hypocondition.

An exact diagnosis of a hypersensitiveness of the vestibular nuclei can, according to Alexander and Brunner, be made from the symptoms of dizziness, spontaneous nystagmus and lengthening of the duration of the caloric nystagmus over a two minute period.

An exact diagnosis of hyperexcitability of the end-organ can likewise be made from a history of vertigo, the occurrence of spontaneous nystagmus, at least at times, and a shortening of the latent period of caloric nystagmus below ten seconds.

In a series of sixty cases in which a diagnosis of commotio cerebri had been made, Brunner found that the eighth nerve was involved in thirty-four cases, or 56.6 per cent. In this group of cases, which is group I of his classification, he found symptoms of concussion of the brain and symptoms in the ears. The symptoms in the ears consist in (1) vestibular vertigo, (2) spontaneous nystagmus, (3) caloric hyperirritability, (4) tinnitus and (5) a normal cochlear apparatus. For these symptoms, Brunner presupposes a lesion in the brain stem, the central vestibular system.

Rhese's [31] ideas on this subject are different than Brunner's. According to him, the demonstration of a vestibular participation after a head injury is based on the demonstration of spontaneous vestibular symptoms (nystagmus, vertigo, past pointing, and falling), and secondly on the reduction or ablation of a reaction after vestibular irritation. In regard to the caloric irritability, he states that one sign of a traumatic vestibular lesion is a varying intensity to stimulation of the two ears, a compensation disturbance based on a lowered irritability of the injured side. If caloric hypo-irritability is combined with normal hearing, the lesion is central. If it is combined with a defect in the hearing, the lesion can be looked for in the end-organ. Hyperirritability of the vestibule which tends to become normal in the lapse of time, he believed, finds its expression on the uninjured side, an evidence of its own increased self stimulation.

Brunner [8] also found a condition of caloric hypo-irritability in some of his cases, group II of his classification, commotio cerebri et auris internae. His idea was that the changes which occur in this group are circulatory disturbances which last a long time. These do not damage the ear function unless they produce changes in the perilymph and endolymph. 1. Such changes in the perilymph only do not have any effect on the function of the vestibule, but do have an effect on the cochlea, producing deafness of the middle ear type. 2. Pathologic changes in the endolymph produce symptoms arising from the vestibule, spontaneous labyrinthine symptoms combined with a limitation or ablation of the caloric function, such as Bárány and Neuman have pointed out.

In my own series of cases, the caloric irritability was carefully tested in thirty patients, in all of whom spontaneous labyrinthine symptoms were present. The thirty cases fall into three general groups.

In group I, consisting of ten cases, the caloric irritability fell within normal limits of time and was of about equal intensity on the two sides in eight cases and unequal in two cases. In most of these patients, however, the Bárány mass irrigation test was performed, by which it is difficult to detect hyperirritability or hypo-irritability, or differences in the irritability of the two ears.

In group II, consisting of eleven cases, the caloric irritability was below normal in one or both ears. The reaction was unequal in intensity in eight cases and equal in three cases.

In group III, consisting of nine cases, the labyrinth was hyperirritable to caloric stimulation. The irritability was unequal in intensity in eight cases, and equal in one.

So of the thirty cases, the irritability of the tested labyrinth, whether normal, hyperirritable or hypo-irritable, was of unequal intensity in

eighteen, and of equal intensity in twelve; and some of the twelve would fall in the other groups had tests been performed by the Kobrak method.

With regard to the associated loss of hearing, I found in group I no loss of hearing in one patient, marked loss of hearing over entire tonal range in both ears in three patients, marked general loss in one ear in one patient, moderate loss in the upper tone range in two patients and a slight loss in the upper range in three patients.

In group II all the patients except two exhibited moderate or marked loss of hearing, mostly in the upper tone range. The remaining two exhibited slight losses of hearing in the upper tone range.

In group III one patient did not show any loss of hearing, one showed marked loss, and the remaining eight showed slight or moderate losses mostly in the upper tonal range.

Only four patients in all those with a hyperirritable labyrinth showed a small loss of hearing and might possibly fall into Brunner's class of cases of commotio cerebri with symptoms of the ears.

From a study of my own material, I am convinced that neither Brunner nor Rhese are correct in assuming that the spontaneous labyrinthine symptoms are caused in the one instance by a hyperirritable condition of the labyrinth, and in the other by a hypo-irritable condition. As I found the labyrinths unequally irritable to caloric stimulation in eighteen of thirty cases (and this figure might be larger were it not for the fact that a number of the group of normal persons were examined only by mass irrigation), I am more inclined to Rhese's view that the appearance of spontaneous labyrinthine symptoms is due to a decompensation between the two unequally irritable sides.

Having taken up the symptomatology of trauma of the head relating to the vestibular portion of the organ of hearing, I will now consider the symptoms relative to a disturbance of the nervus cochlearis and its end-organ, the cochlea. My method of examination of the hearing is to test out each ear for the whisper, the other ear being closed by a moist finger. The hearing for the watch is tested in the same manner. The Weber reaction was usually taken in the early stages of this work, but it was found to have so little importance that it was not always obtained. The Rinne and Schwabach tests were always made, and I would say in this connection that a great deal of care must be exercised in making these tests to avoid the phenomenon of fatigue on the part of the patient. This test was always made by holding the stem of a *C* 1 (265 double vibrations) fork against the mastoid process in the vicinity of the antrum, each ear being tested separately. The stem of the fork is placed on the bone, removed and then replaced until the patient no longer hears it, and the same technic is employed on the mastoid of the examiner. In this way I feel certain that the phenome-

non of fatigue for the bone conduction of the tone was avoided. For the lower tone limits a large weighted Dench fork of 32 double vibrations was used. The upper limits were usually worked out on the audiometer, but when this was not available, the *C* 5 fork and the Galton whistle were used.

In working out the loss of hearing, and especially in studying the changes in the hearing from time to time, the audiometer was found to be of great assistance. The same results could undoubtedly be obtained by examination of the patient with a continuous series of tuning forks, but this method of examination is tedious to both patient and examiner. I believe that with the audiometer I have been able to pick up traumatic defects of hearing which would have escaped me had I resorted only to the other tests for hearing. The Western Electric no. 2*A* audiometer was used.

Tinnitus was complained of only seven times in the entire forty-two cases, and apparently did not have any relation to the severity of the trauma or to the degree of the resulting deafness.

The Rinne reaction was positive in thirty-five cases, negative in one case and not taken in six cases. This rather constant observation agrees with the reports of Brunner,[8] Mauthner,[28] Mygind [2] and Rhese.[38] Passow [1] said that the Rinne reaction is always positive, as may be expected from the shortened bone conduction.

The bone conduction was shortened in twenty-four cases, normal in six cases and not taken in twelve cases. There seemed to be considerable doubt in Brunner's mind as to the existence of a shortened bone conduction, for he found the bone conduction normal in all his early cases except two in which it later became normal. He did not consider bone conduction shortened, however, unless it was more than six seconds short. This, I believe, is extending the limits of error too far, if one is careful to avoid the phenomenon of fatigue on the part of the patient. Nor did Mygind [2] see any shortening of bone conduction in his patients. On the other hand, Mauthner,[28] Rhese [38] and Passow [1] always found a shortened bone condition, and Passow stated that this is the cause of the constantly present positive Rinne reaction. Wanner and Gudden [39] found the bone conduction shortened in cases of so-called traumatic neurosis in which the hearing was normal. Hasslauer [40] found a shortening of bone conduction in twenty-two cases of injuries to the

38. Rhese: Beiträge zur Kenntniss der Beteiligung des inneren Ohres nach Kopferschütterungen, Deutsche med. Wchnschr. **32**:625, 1906.

39. Wanner and Gudden: Die Schalleitung der Schädelknochen bei Erkrankungen des Gehirns und seiner Häute, Neurol. Centralbl. **19**:883, 1900.

40. Hasslauer: Die Verwertung des Schwabachschen Versuches bei der Diagnose intrakranieller Veränderungen, München. med. Wchnschr., 1910, p. 470.

head, some mild and some severe. Herzog [41] took exception to the observations of Hasslauer and Wanner and Gudden that shortened bone conduction with normal hearing is a sign of trauma of the head, because, as he said, shortened bone conduction is a sign of disease of the inner ear, and the foregoing authors did not prove that the hearing was normal. To determine this every possible method of examination should be used, particularly at the upper tone limits, and Herzog believes that the determination of the upper tone limits with the Galton whistle is unreliable, because many people who have poor hearing for a *C* 4 or *C* 5 fork hear the whistle to the end.

I can heartily endorse this view of Herzog. A shortening of bone conduction means disease of the inner ear, and it is always associated with some loss of hearing in the upper tone range. Furthermore, the testing of the upper tone range solely with a Galton whistle, or even roughly with the *C* 4 and *C* 5 forks, is insufficient, as the following case will illustrate. The audiometer offers a quick and accurate method of testing the entire tone range in steps of one octave.

Case 9.—*History.*—John Kurlinsky, aged 52, was struck in the left parietal region, close to the midline, on Oct. 22, 1926, by a piece of pipe which fell a distance of from 45 to 50 feet. He was unconscious and in a dazed condition until he reached the hospital. Bleeding from nose, mouth or ears was not noted. During the next two weeks in the hospital, he was in a more or less dazed condition, and his memory for events in the hospital was poor except for isolated facts. He remained in bed in the hospital for seventeen days. Headache was present every day while he was there. As soon as he regained consciousness and raised his head, he was dizzy. About three days after his injury, he discovered that he could not read the print in the Sunday paper.

By Feb. 1, 1927, the headache was gradually disappearing, but it returned at times and was referred to the healed scar of his original wound. His vertigo was also gradually disappearing but returned at times, especially when he made sudden movements. He became dizzy when he bent forward and tried to rise from a chair. His vision was still poor but it was inproving. A buzzing sound developed in his left ear while he was still in the hospital, and he did not think that he heard as well with the left ear as with the right ear since the accident.

Examination.—Spontaneous Phenomena: The Romberg sign was negative. The forward gait with the eyes closed was normal. He stood well on either foot with the eyes closed. There was a slight spontaneous rotary-horizontal nystagmus to the left, but no spontaneous past pointing or deviation.

Eyes: The vision of each eye was 20/50. This could be corrected to 20/20 by proper lens, and with the addition of the proper presbyopic correction, he could read the finest Snellen type at the near point. The pupils were small, round and equal and they reacted to light and in accommodation. There were no palsies of ocular muscles. The media were clear and the fundi were negative.

Nose and Throat: Observations in the nose were negative, and the tonsils were not enlarged or infected.

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41. Herzog: Kritisches zur Verkürzung der Knochenleitung bei normalen Gehör, München. med. Wchnschr., 1913, p. 18.

Ears: Both external canals were clear. Both drums showed slight retraction. Redness was not present in either drum. The light cone was normal on both sides. There were not any scars or perforations. Tests of the hearing yielded the following results:

Tests of Hearing	Right Ear	Left Ear
Whisper	At 20 feet plus	At 8 feet
Watch	At 15 inches	On contact
Rinne	Positive 5; 13	Positive 5; 13
C (32 double vibrations)	Hearing	Hearing
Galton whistle	To end	To end
Schwabach	Six seconds short	Eight seconds short

Caloric Tests.—Right Ear: A mass irrigation of water at 20 C. produced in eighty-two seconds a rotary-horizontal nystagmus to the left, past pointing to the right with both arms and considerable vertigo.

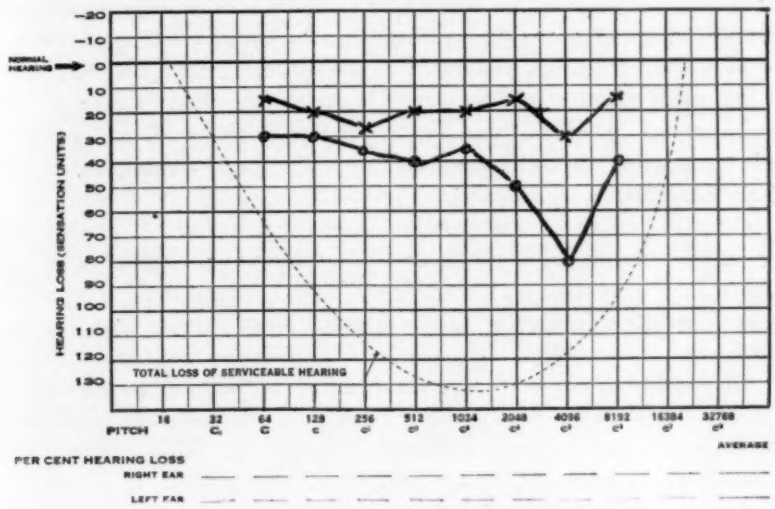

Chart 5 (case 9).—Audiograms of both ears.

Left Ear: A mass irrigation of water at 20 C. produced in forty-two seconds a rotary-horizontal nystagmus to the right, and past pointing to the left with both arms. The vertigo was marked.

In case 9, one sees the absolute disproportion between the hearing for the Galton whistle, on the one hand, and the hearing for the tone of 4,096 double vibrations, on the other. If one simply tested out the upper limit with the Galton whistle one would say that it was normal. The man in this case had a traumatic disease of the inner ear, more marked in the left ear, and more pronounced at the level of *C* 5 or 4,096 double vibrations, above which point his hearing was again improving, so that when the level of the tone of the Galton whistle was reached, he was able to hear it to the end.

Deafness, the chief sign of damage to the cochlea or to the cochlear nerve was complained of by only fourteen patients of forty-two during the taking of the histories. It was never suggested to the patient during this portion of the examination by the asking of leading questions. However, a traumatic deafness caused by the injury of the head was uncovered during the examination in thirty-one of the forty-two cases of the series. In four of the remaining eleven cases, the examination of the hearing was not accurate enough to determine whether any defect existed or not.

In seven of my patients, the hearing after the accident was normal. Of these seven, three had rather severe accidents, in two of which bleeding occurred from both ears. One of the patients, a woman with normal hearing, had her head jammed between the doors of a street car and presented later the vestibular symptoms of vertigo, spontaneous nystagmus, spontaneous past pointing and a hyperirritable labyrinth on one side, with a normal irritability of the other. This is the only case in all my series which might be placed in Brunner's class of cases of commotio cerebri in which there are symptoms in the ears, and which, he believes, are due to vasomotor disturbances in the vestibular nuclei region of the medulla.

Complete deafness was present in one ear in patient 1. In the other ear of this patient there was a loss of hearing in the upper tone range (chart 1). This man undoubtedly suffered a transverse fracture of the right pyramid, together with considerable hemorrhage into the basal coil of the left cochlea.

In another group of ten patients, a loss of hearing was found over the entire tone range. Of these ten patients, in eight there was bilateral involvement, and in two unilateral involvement. The following case record may be regarded as a prototype of this group:

CASE 10.—*History.*—Gus Preiss, aged 39, while working for a packing company in April, 1926, had his head caught, as if in a vice, between the chain and the wheel of the conveyor chain. The head was caught between the right ear and the left temple and was held in this position for some time until one of the spokes of the wheel broke and released it. This was at about 2 or 3 o'clock in the afternoon. Immediately after this he did not remember anything until he was taken to the plant physician. He stated that he stayed on the job the rest of the afternoon, but said that he had been off the job at various times since the accident because of headaches. He said that the headaches came on when he looked down and were associated with attacks of dizziness. These symptoms appeared soon after the accident and persisted up to the time of the examination. Bleeding from the ears, nose or mouth was not observed.

By May 6, 1927, he had headaches several times daily for short periods, lasting, perhaps, only from two to five minutes, and they were associated with dizziness. They were brought on especially by bending down.

Examination.—The patient had a saffron yellow complexion. He stated that he had recently had yellow jaundice.

Spontaneous Phenomena: Spontaneous rotary-horizontal nystagmus was observed to the right and to the left in the end positions, much more marked in the left end position. There was a spontaneous past pointing to the left with both arms, shoulders down; that of the left was double that of the right. The Romberg reaction was negative, and deviation in the forward gait was not present with the eyes closed.

Ears: Both external canals were clear and normal. The right tympanic membrane showed retraction, loss of luster and absence of the light cone. The left drum was fairly normal, except for a slight shortening of the light cone.

Nose and Throat: The nose was normal except for a deviation of the septum to the left, with a sharp spur in the lower portion. The tonsils were of normal size, and the pillars were not injected. The crypts of the right tonsil contained pus; those of the left did not.

Tests of the hearing yielded the following results:

Tests of Hearing	Right Ear	Left Ear
Whisper	At 8 feet	At 8 feet
Watch	At 8 inches	At 8 inches
Weber	Not referred	
Rinne	Positive 5; 11	Positive 4; 11
Schwabach.....	Two seconds short	Thirteen seconds short
C (32 double vibrations)..	Normal hearing	Normal hearing
C (64 double vibrations)..	Hearing	Hearing
C (4,096 double vibrations)	Hearing (struck hard)	Hearing (struck hard)

Caloric Reactions.—Right Ear: Fifteen cubic centimeters of water at 15 C. produced in forty-five seconds a horizontal-rotary nystagmus to the left of small amplitude, lasting seventy seconds. Deviation of the right arm was not elicited in the pointing test, and there was only a moderate deviation of the left arm to the left. Slight vertigo was present.

Left Ear: Fifteen cubic centimeters of water at 15 C. produced in thirty seconds a slight slow rotary-horizontal nystagmus to the right, lasting ninety seconds, with marked past pointing to the left with both arms. Only slight vertigo was noted.

This man apparently did not suffer a severe injury. At any rate, he was able to resume his work for the rest of the day. His complaints were mainly of headache and vertigo, coming on in attacks of short duration, and aggravated by bending movements of the head. His spontaneous labyrinthine symptoms were those arising from both vestibular regions, but they were more marked on the left side. The loss of hearing was bilateral, being marked over the entire lower and middle tone range, and complete for the upper tone range.

In another patient in this group of ten, the loss of hearing was largely unilateral, and due, I think, to a longitudinal fracture of the left petrous bone, with an infected hematotympanum which resulted in a chronic mastoiditis.

Case 11.—*History.*—C. A. Emburr, aged 44, was riding in an automobile which was run into on July 4, 1924. He did not remember anything from immediately after the accident until he picked himself up in the bottom of the car. He had

bruises on the left side of his head and on his left knee. He said that his left ear began to ring immediately, and he had not heard anything with it since. It did not bleed but began to discharge on the day after the accident. It discharged continuously for six weeks, and thereafter from time to time until his examination. He stated that he had more or less constant pain in the ear.

Examination.—Ears: The right external canal was clear. The right drum was lusterless and there was a moderate degree of retraction. The light cone was shortened, and the short process of the hammer was prominent.

The left external canal contained a mucopurulent secretion in its inner end. There was a large defect of the drum, involving the entire anterior portion of the drum with the exception of Shrapnell's membrane and a small rim at the posterior margo.

Nose and Throat: A high deviation of the septum to the left was observed. Free pus was seen on both sides of the nose coming from the middle meati. The tonsils were small and not infected.

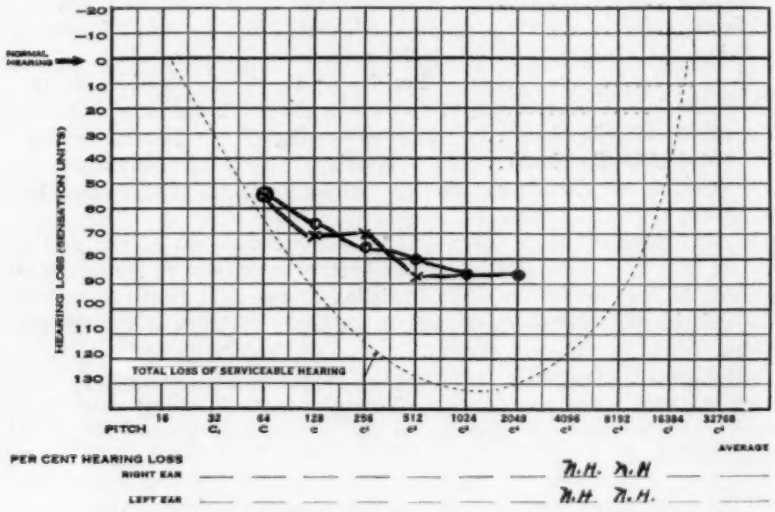

Chart 6 (case 10).—Audiograms of both ears.

Tests of the hearing revealed the following results:

Test of Hearing	Right Ear	Left Ear
Whisper	At 20 feet	At 1 foot (badly)
Conversation		At 8 feet
Watch	At 8 inches	Normal hearing
Rinne	Positive	Negative
Schwabach	Normal	Five seconds short
Weber		To left

Vestibular Tests.—Spontaneous nystagmus was not noted. The Romberg reaction was negative. Because of the discharging ear, the caloric tests were not used, but the turning tests gave normal responses on both sides.

X-Ray Examination.—The left mastoid showed definite clouding and haziness of the cell structures throughout. Evidence of skull fracture could not be made out on the frontal and lateral views of the head.

This case history is presented because I believe that the patient sustained a longitudinal fracture of the left temporal bone. He experienced immediate tinnitus and loss of hearing in the left ear, followed by a discharge from it on the following day. The x-ray observations on fractures in this area are notoriously unreliable, and, therefore, cannot be relied on for corroborative evidence.

The three audiograms in charts 8, 9 and 10 illustrate the fact that when the injury results in loss of hearing in the lower and middle tone range, much of the loss may be due to an involvement of the middle ear, and improvement may be expected ultimately. On the other hand, the loss in the upper tone range, which, I believe, is always due to damage in the basil coil of the cochlea, is apt to be permanent or even become worse after the lapse of time.

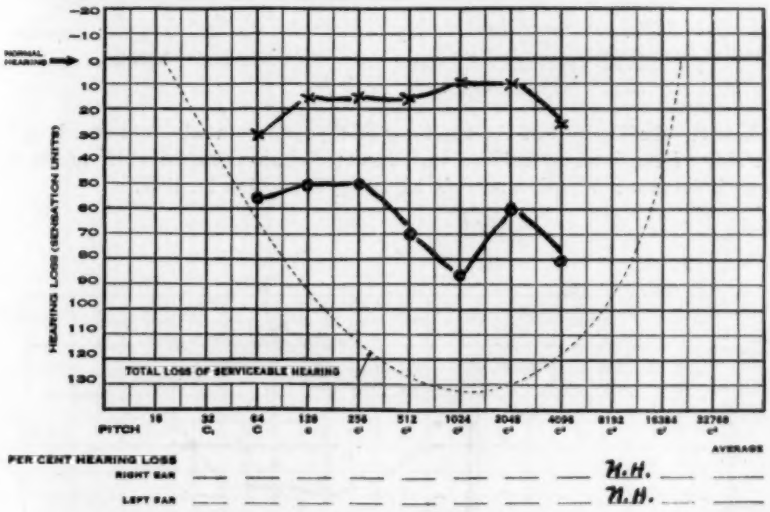

Chart 7 (case 11).—Audiograms of both ears.

Chart 8 is the audiogram of a man who was struck on the right side of the head by a large steel hook which was used in raising and lowering buckets from a ditch. He did not become unconscious, but he bled from the right ear. He had a chronic purulent otitis media of the left ear of long duration. The injury occurred about Jan. 15, 1927. The lower and middle tone range improved, while the defect in the upper tone range remained the same, as may be seen in the chart.

Charts 9 and 10 are the audiograms of a young woman who was thrown from an automobile to the highway. She lost consciousness, but did not bleed from either ear. She had marked vertigo and spontaneous nystagmus to the right side during the first week after the accident. Spontaneous past pointing and falling to the left were also

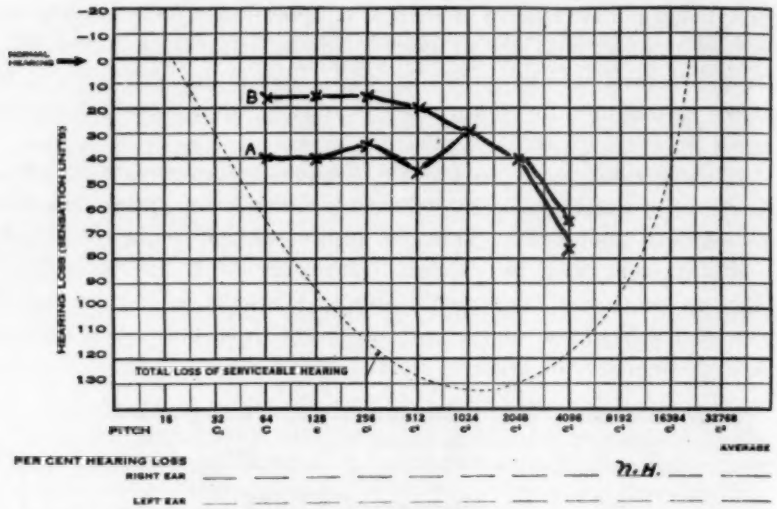

Chart 8.—Audiograms of the right ear of a young man who was struck on the right side of the head by a large steel hook; chronic purulent otitis media was present in left ear: *A*, taken on Feb. 15, 1927; *B*, on May 11, 1927.

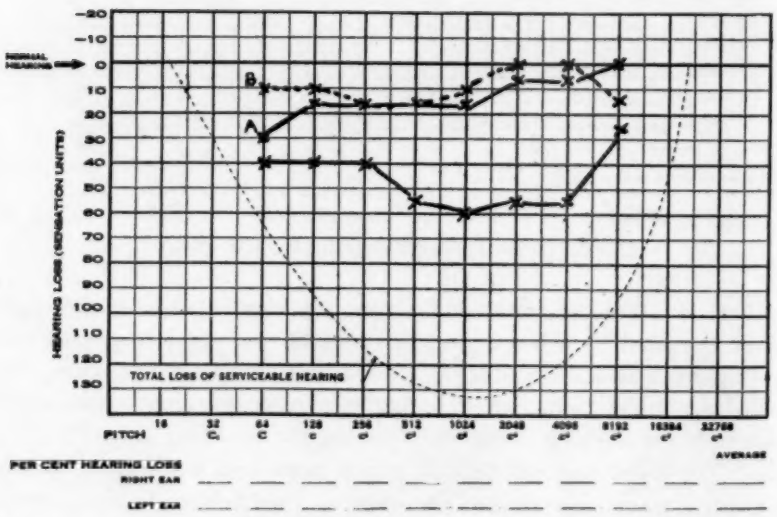

Chart 9.—Audiograms of the right ear of a young woman who was thrown from an automobile to the highway; lowest curve, taken on March 13, 1926; *A*, on Dec. 18, 1926; *B*, on Feb. 10, 1927.

present. The loss of hearing, when she was examined two weeks after the accident, was marked in the right ear over the lower and middle tone range, and in the left ear over the entire tone range. In the course of eight months the hearing in the right ear returned to normal, while that of the left became a little worse, particularly in the upper tone range. In this connection, it is interesting to note that the bone conduction was slightly increased on the right ear after eight months, while it was shortened seven seconds on the left. The original loss in the right ear was due undoubtedly to changes in the middle ear, while that of the left ear was due to extensive hemorrhage in the inner ear. Chart 9 shows the improvement in the entire range of the right ear. Chart 10 shows some slight improvement in the lower tone range of the left ear,

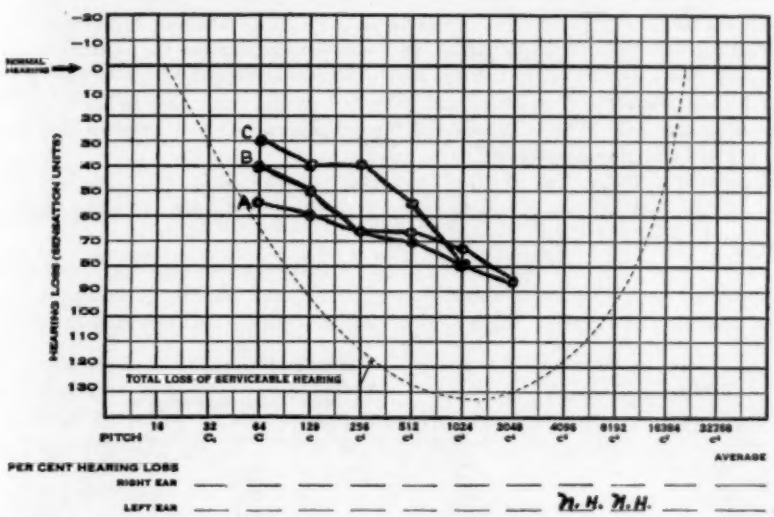

Chart 10 (same patient as in chart 9).—Audiograms of left ear; *A*, taken on March 13, 1926; *B*, on Dec. 18, 1926; *C*, on Feb. 10, 1927.

while the middle and upper tone ranges do not show any improvement in eleven months' time. The last audiometric examination, made two and one-half years after the injury, showed a still further improvement in the lower tone range of the left ear while the middle and upper tone defects remained unchanged.

In the next group of ten patients, the loss of hearing affected the entire upper tonal range while the lower and middle range was affected little or not at all. In five of the patients, the defect in the hearing was bilateral, and in the other five it was unilateral. An example of this group is found in the following record:

Case 12.—*History.*—Henry Heinrich, aged 45, fell from a scaffold, from 10 to 12 feet high, to the ground, striking the ground with his head, on Nov. 11, 1926. He was taken to the hospital where he remained for four weeks. For the first

two weeks, he was in a state of coma. After going home for a month, he returned to the hospital for four weeks longer. There was not any history of bleeding from the ears, but he bled from the mouth. He noticed vertigo when he got out of bed for the first time, three weeks after his accident. This condition was definitely not a rotary vertigo, but rather an up and down error of sensation. The vertigo was never constant, but came on in attacks. He had a bad double vision after his injury, for which he began wearing prisms.

On Aug. 2, 1927, the vertigo had been increasing for two weeks. He had two or three attacks a day, which lasted at times only a few minutes, and at others an hour. During the attacks he felt as if he was drunk. A sense of rotation was not noted during the attack. He had headache occasionally along with the vertigo. The vertigo apparently became worse after meals, after he had been up on a roof or out in the sun. Vertigo also came on after he bent his head back or when he looked down, but this was momentary. He still had double vision occasionally, but this was improving.

Examination.—Spontaneous Phenomena: A spontaneous rotary-horizontal nystagmus was present to the right; this was increased after the head movement test, and then a nystagmus to the left also appeared, accompanied by vertigo. There was a spontaneous past pointing and deviation of the right arm to the right, but none with the left. The Romberg reaction was normal. The forward and backward gaits with the eyes closed were normal.

Eyes: The vision was 20/20 in each eye. Ocular palsies were not observed. Diplopia was not present except in the end position to the left. The pupils were equal and reacted to light.

Ears: Both external canals were clear. Both drums showed slight retraction. There was hyperemia of Shrapnell's membrane and of the posterior superior canal wall near the drum. Tests of the hearing revealed the following results:

Tests of Hearing	Right Ear	Left Ear
Whisper	At 1½ feet	At 3 feet
Watch	Normal hearing (bone or air)	Normal hearing (bone or air)
Weber	Referred to right	
Rinne	Positive 11; 38	Positive 17; 33
Schwabach	Five seconds short	Two seconds short

Caloric Test.—Right Ear: Five cubic centimeters of water at 15 C. produced in twenty-eight seconds a rotary-horizontal nystagmus to the left, lasting twenty-five seconds in position I only, and no vertigo.

Left Ear: Five cubic centimeters of water at 15 C. produced in thirty-six seconds a small rotary-horizontal nystagmus to the right, lasting fifty seconds, and no vertigo. Both labyrinths were slightly hypo-irritable.

Finally, in a group of twelve patients, I found a loss of hearing revealed on the audiogram which is hard to explain. This loss was indicated by a more or less isolated notch in the region of the *C* 5 (4,096 double vibrations) tone. The hearing was fairly normal below this point and again rose to approximately normal above this point. In fact, in the audiogram of patient 9, chart 5, the loss of hearing at this point, which is marked in the left ear, improved again at the tone of *C* 6 (8,192 double vibrations), and the patient was able to hear the Galton

whistle to 0 in each ear. This notch can also be seen in the audiogram of patient 5, chart 2. This notch was found to be bilateral in eight cases and unilateral in four. The best explanation which I can offer for the phenomenon is that there was a more or less isolated hemorrhage in the basal coil of the cochlea.

The twelve patients might easily pass for persons with normal hearing if they were examined by the usual tests for hearing, but the examination with the audiometer always reveals a loss of hearing or tone gap at this particular point.

SUMMARY OF CASES

I found some loss of hearing in thirty-one of my series of forty-two patients and normal hearing in seven; account is not taken of the

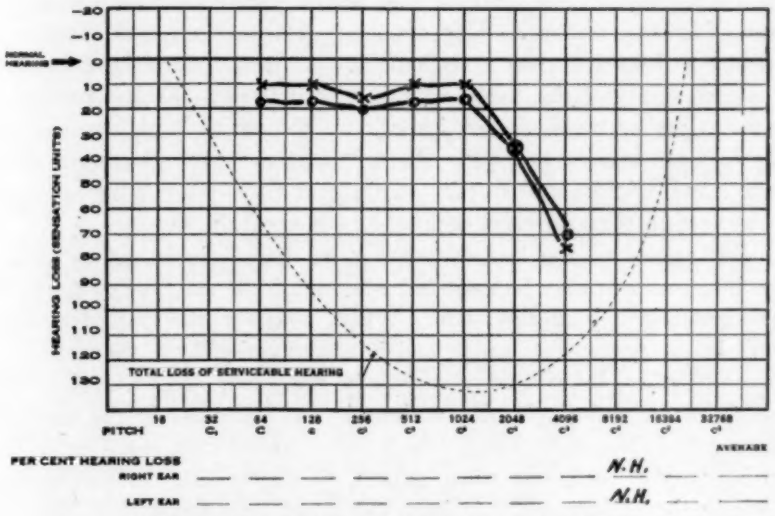

Chart 11 (case 12).—Audiograms of both ears.

remaining four patients because the tests of the hearing were incomplete. Of the thirty-one patients, twenty-eight had symptoms referable to the vestibular apparatus.

In the classification of injuries of the head from the standpoint of otologic damage, I would be inclined to use the following classification: (1) transverse fracture of the pyramid; (2) longitudinal fracture of the petrous temporal bone with commotio labyrinthi, and (3) commotio cerebri with commotio labyrinthi.

It is to be understood in this connection that by commotio labyrinthi, I have reference to actual damage to the intralabyrinthine structures by hemorrhage, which later either is absorbed or causes atrophy and degeneration of the nervous elements.

COURSE AND PROGNOSIS

If the vertigo, so frequently complained of by the patient with trauma of the head, can be shown to be of true labyrinthine origin, little improvement may be expected over a long period. Brunner reported ten cases which could be followed for a long time, and of these, six presented an unchanged picture for ten years.

In the traumatic neuroses which subsequently develop in these cases of injury to the head, the question of vertigo is of prime importance. The vertigo complained of by these patients is of labyrinthine origin if characterized by a sense of rotation, if produced or aggravated by bending the head or, according to Brunner, if accompanied by caloric hyperirritability, even though not of rotary character.

The neurotic type of vertigo improves under treatment and monetary settlement, but the vestibular vertigo reappears from time to time for years after the trauma. This type of vertigo does not show any tendency to improve after a settlement. Alt was quoted by Brunner [8] as having said that the vertigo arising from lesions of the vestibular apparatus makes much deeper inroads on the patient's ability to work than does his loss of hearing. Rhese,[38] Ruttin [42] and Fletcher [43] were of the same opinion, and Ruttin went so far as to place a higher value on the vertigo than on total deafness of one ear.

With regard to the loss of hearing in these injuries of the head, some improvement may be expected when a middle ear type of deafness is encountered in young persons. When, however, the loss of hearing occurs in the upper tone range and is accompanied by a shortening of bone conduction, improvement cannot be expected. In fact, when one remembers the pathologic observations in the inner ear in the cases of Manasse and Theodore, cases which came to autopsy years after the injuries to the head, one must expect, whenever the perilymph, and especially the endolymph, has been disturbed and replaced by hemorrhage, that a gradually progressive type of deafness will ensue.

The prognosis of transverse fractures of the pyramid is bad, because in many cases the patients die shortly after the injury. If they recover, the prognosis for hearing is bad, because it is irrevocably lost. The intensive vestibular symptoms which were present in the first few weeks gradually subside. Patients who recover from a transverse fracture of the pyramid are not in any special danger from late meningeal infections from the middle ear, because it is only rarely opened by this type of fracture.

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42. Ruttin, E.: Ueber frische traumatische Läsionen des Labyrinth, Monatschr. f. Ohrenh. **46**:475, 1912.

43. Fletcher, Harold: Determination of Disability as to Loss of Hearing and the Importance of Vertigo in Industrial Accident Cases, J. A. M. A. **79**:529 (Aug. 12) 1922.

The longitudinal fractures of the temporal bone, on the other hand, which course through the tegmen tympani or tegmen antri or through the canalis musculotubarius, rupturing the drum and fracturing the external auditory canal, behave differently. Because of the well known tendency of labyrinthine fractures to close by fibrous, instead of bony, union, persons with this type of fracture are in continual danger of late meningeal infection from the middle ear. Lange [44] reported a case, in which the patient died of an abscess of the brain two years after an injury in which the labyrinthine capsule was fractured. He found that the fracture had healed by connective tissue and bone, but excess callous formation was not found in the labyrinthine spaces, as described by Manasse [24] and Nager.

CONCLUSIONS

1. Patients with injuries of the head must be examined as soon after the injury as possible, and again at regular intervals over a considerable period.

2. The severity of the injury does not bear any direct relation to the development or degree of cochlear and vestibular symptoms.

3. Most of the injuries to the head in civil life are caused by a broadly acting force which compresses the skull, whether it fractures it or not. This compressing force results in damage to the brain, to the cerebrospinal fluid and to the vascular system of the blood, as well as to the skull.

4. With regard to the skull, the compressing force of the injury finds its greatest expression at the base, because of the more or less unequal strength of its constituent parts; and the middle fossa, being weaker than the anterior or posterior fossae, is most often affected. The pyramid, weakly attached in the middle fossa, takes the brunt of the injury to the base and is frequently damaged.

5. Fractures of the temporal bone are divided into transverse fracture, longitudinal fracture and avulsion of the tip of the petrous temporal bone. Of these, the longitudinal fractures are the most numerous, do not damage the labyrinth by fracture but by the concomitant concussion and usually involve the middle ear and external canal directly by fracture. The transverse fractures are more rare, cross the pyramid at right angles and completely destroy both the vestibule and the cochlea. Avulsion of the tip of the petrous bone is relatively rare.

6. The damage to the brain is caused by actual compression of the brain beneath the point of impact and at a point directly contrecoup. The damage done by the cerebrospinal fluid is caused by the compression of the lateral ventricles which sets the fluid in motion to expend its

44. Lange (reference 19, second part).

force in a whirlpool action in the fourth ventricle. The damage to the blood vascular system consists in a state of traumatic paralysis of the vasoconstrictors with resulting stasis and prestatic circulation in the brain tissue, particularly in the central vestibular area, and probably also in the labyrinth.

7. Within the temporal bone the salient features of all patients who died and who came to autopsy were hemorrhages. The intralabyrinthine hemorrhages are always perilymphatic, unless the capsule of the labyrinth is fractured, in which case they can also be endolymphatic. The most frequently affected region in these intralabyrinthine hemorrhages is the scala tympani in the vicinity of the round window. The nerves can be either torn or damaged by pressure from hemorrhage, before their entrance into the pyramid, within the porus acusticus internus or in the narrow bone canals leading to the end-organ.

8. The pathologic changes in patients dying years after injury to the head are atrophy of nerve fibers, atrophy of Corti's organ, more marked in the basal coil, and complete or partial filling of the inner ear spaces and canals with hyaline connective tissue and bone.

9. The results of experimentation on animals show that the effects of mild injuries are the same, in lesser degree, as those found at autopsy in the temporal bones of human beings, namely, hemorrhages in the inner ear, more marked in the basal coil of the cochlea and in the region of the round window, always in the perilymph spaces and never in the endolymph spaces. Degenerative changes are seen also in the nuclear territory of the eighth nerve in the floor of the fourth ventricle, affecting mainly the small cells of the nervus triangularis, the nucleus of von Bechterew, the tuberculum acusticum and the posterior corpora quadrigemina. These changes are probably due mostly to vasomotor disturbances in this section producing a condition of stasis with subsequent destruction of tissue.

10. Hemorrhage from one or both ears occurred in nine cases in my series, and I hold that this constitutes almost indisputable evidence of a longitudinal fracture of the temporal bone. This bleeding from the ear, however, does not mean that great damage to its function will necessarily ensue.

11. Vertigo is one of the cardinal symptoms of injury of the vestibular system. It is vestibular in origin if it has a rotary quality, if it comes on in attacks, accompanied by nystagmus or if it is produced by bending movements of the head. Other types of vertigo after injuries to the head are apt to be neurotic, especially if they are constantly present after the first two weeks, or if they are accompanied by severe nausea, vomiting and great mental excitement. I am always

strongly suspicious of a neurosis, if the patient, after the first week or two, complains of constant headache and vertigo.

12. Spontaneous nystagmus is another cardinal symptom of vestibular injury. It is unilateral or bilateral, and if bilateral, it is more marked to one side. It is always rotary-horizontal in character. I believe that it is due to a decompensation between the two vestibular systems. Bilateral nystagmus to the two sides is often encountered in normal people, but it is always of the pure horizontal type, always equal in intensity, not associated with vertigo and never influenced by the head movements test. I cannot formulate any rule for the direction of the vestibular nystagmus in my cases.

13. Disturbances in the pointing reaction were present in twenty-eight of forty-two cases and they constitute a part of the spontaneous vestibular symptoms which occur after damage to the vestibular system. No great reliance can be placed on them in deciding which side is involved. The same holds true for the falling and Romberg reactions.

14. The irritability of the labyrinth to caloric stimulation was carefully studied in thirty cases, all of which presented spontaneous labyrinthine symptoms. Normal reactions were obtained in ten cases, hypo-irritability in eleven cases and hyperirritability in nine cases. The comparative irritability of the two sides was equal in twelve cases and unequal in eighteen cases. I believe that a difference in the irritability of the two sides, in other words, a decompensation between the two labyrinths, is of far more importance in the production of the spontaneous labyrinthine symptoms than is hyperirritability or hypo-irritability per se.

15. The Rinne reaction was positive in thirty-five cases, negative in one case and not recorded in six cases. Bone conduction was shortened in twenty-four cases, normal in six and not recorded in twelve. Traumatic deafness, caused by injury of the head was found in thirty-one of forty-two cases. Complete deafness in one ear was present in one case. In a large proportion of the cases the defect in the hearing was bilateral, and a large number of the cases showed the upper tone range more affected than the lower and middle ranges. Of the thirty-one patients with defective hearing, twenty-eight had symptoms referable to the vestibular apparatus.

SOME ACHIEVEMENTS IN AMERICAN LARYNGOLOGY AND RHINOLOGY *

D. BRYSON DELAVAN, M.D.
NEW YORK

The American Laryngological Association commemorates this year the most significant and interesting event in its history, its fiftieth anniversary. As its representative, I bid you welcome.

First, let me extend the hearty welcome of the association as well as of myself to our distinguished guest of honor, and my long time and highly valued friend, Sir St. Clair Thomson. No greater compliment could be paid us than the presence and support of him who is the acknowledged leader of laryngology of the old world.

In the name of the association, I welcome the younger members, especially those recently admitted.

And, let me say, this is no ordinary greeting, for we welcome you to the jubilee of an institution, the first of its kind, the oldest among those others which have tardily followed its inspiring example. Always in the advance, for half a century of continuous usefulness it has sturdily upheld the high ethical and scientific standards set by those distinguished men who were its founders and whose mantle of energy and genius has ever since rested on their successors. We do well to waive undue reticence and celebrate the occasion with befitting eulogy, and with fair regard to justice and truth.

It is not without emotion that I stand before you today, overwhelmed by a flood of memories. Especially do I recall my entrance into the association and the friends who were its members earlier than myself. But one of them remains—Dr. French, well deserving the Honorary Fellowship he is about to receive. Of those active at the time of my presidency, in 1894, eight survive, namely, Birkett, Bryan, Coolidge, French, Rice, Swain, Wagner, Watson and myself. Four of us are present at this meeting.

Thoroughly familiar with the earliest beginnings of the association through my relations with Dr. Lefferts, one of its most active founders, and made still more intimate three years after its birth, by seven years of service as its secretary, my association with it has been a life-long satisfaction. No other medical interest of its kind has equaled it. For it I have labored with unremitting enthusiasm. From it I have drawn uplifting inspirations. In it I have made many of my best and warmest

* The Presidential Address, delivered before the Fiftieth Annual Congress of the American Laryngological Association, Washington, D. C., May 1-3, 1928.

and most delightful friends. My interest in it has strengthened step by step with its steady growth.

I cannot begin my remarks more fittingly than to quote the opening of the address of Dr. Logan who in 1910, filling the presidential chair, said:

"It is something, I think it is much, to be able to escape even for a brief space the heavy atmosphere of the sordid plain in which we pass our work-a-day lives and get a few whiffs of the ozone that comes from the heights of impersonal, unselfish endeavor." In such spirit, let us celebrate this anniversary.

Just thirty-four years ago I also had the honor to preside. My opening remarks at that time may interest you. There is need for little change in them today.

"As president, the option is accorded me of inaugurating our exercises with the time-honored ceremony of a presidential address. While some have held the value of the presidential address in question, I believe that it should be considered, in like manner with our annual dinners, a very desirable, if not an indispensable part of each yearly gathering, according opportunity to call attention to ideas and suggestions of timely interest which could hardly otherwise be presented, and to record and study events of historical value in the career of the association.

"Surely the experience gained in sixteen years" (we can now say fifty years) "should give sufficient light to illumine the present and enable us to look forward somewhat into the future. In trying to forecast the future, it is eminently right, therefore, that we should indulge in retrospect, particularly for the benefit of any who may have recently come among us, and to whom our early history may be unknown, for the history of the association is almost the history of laryngology itself."

By an interesting coincidence, the year of our celebration falls on the hundredth anniversary of the invention of that instrument of perennial world wide fame, the venerable progenitor of an ever-increasing army of modified descendants throughout the world, the tonsillotome, devised by the distinguished Dr. Philip Physick of Philadelphia in 1828, twenty years before the birth of laryngology in New York.

HORACE GREEN, "FATHER OF LARYNGOLOGY"

No time could be more appropriate than this for a review of the influences which the United States has exerted in the domain of laryngology. What, it may be asked, has our country, what has this great national society whose semicentennial we are now celebrating, done for the advancement of our specialty? The question is eloquently answered by the record of our achievements.

Thus, it was through the heroic efforts of Horace Green, in 1846, that, by the promulgation of his discovery of the tolerance of the larynx to the presence of a foreign body and the application of this discovery to the practical uses of clinical medicine, the science was founded. He well deserved the title universally accorded him of "Father of Laryngology." The contributions of Horace Green's contemporary, Gurdon Buck, rightfully won for him the distinction of "Father of Intra-Laryngeal Surgery." Following the introduction of the laryngoscope in Europe, Solis-Cohen of Philadelphia established organized instruction in laryngology in America, and in 1872 published the first complete system of this subject in either this country or in Europe. In 1873, Clinton Wagner organized the first special hospital in this country; but he far more greatly distinguished himself by founding the New York Laryngological Society in 1873, the first society of laryngology in the world. The more than ten years of its existence, part of which I shared, were important factors in the progress of laryngology, for it was the arena in which was exploited the pioneer work of Bosworth, Jarvis, Beverley Robinson, Elsberg, Lefferts, Lincoln, Asch and others of the brilliant group which composed its membership. One of its most memorable meetings was that at which, in 1882, under the presiding of Dr. Lincoln, the society entertained Sir Morell Mackenzie.

In 1873, Dr. Luis F. Sass of New York created a revolution in the treatment of the upper air passages by the introduction of the spray apparatus, and Lefferts began his perfected system for the instruction in laryngology of undergraduates. Almost simultaneously Bosworth, acting on the suggestion made many years before by Catlin, the American missionary to the Indians, and, subsequently, by Clinton Wagner, demonstrated the evils of nasal obstruction and advanced principles for their relief which rightly won for him the title of "Father of Rhinology." Other early contributors of valuable original work in rhinology were Harrison Allen, John N. Mackenzie, Ernest Shurly, Bryon, Daly and Roe.

The pioneer work of Bryan in the study of the anatomy and pathology of the nasal sinuses and the highly original methods devised by Roe for the repair of deformities and injuries of the nasal framework long antedated similar work abroad. The same may be said of Bosworth, Asch, and Emil Mayer in relation to the surgical procedures on the nasal septum.

It was French who in the early eighties perfected the art of laryngeal photography, and Abbe who years later was the first to demonstrate the value of radium for the cure of laryngeal growths.

Another name worthy of the highest rank stands forth prominently. Joseph O'Dwyer, supplementing the discovery of Green of the tolerance

of the larynx to the presence of a foreign body, established the great physiologic principle of the tolerance of the larynx to the *continued* presence of a foreign body and acting on this principle devised methods from which have been developed some of the most important advances of the present day. Thus he perfected intubation for the relief of acute laryngeal obstruction; he was the first to propose the effective method of curing chronic cicatricial obstructions by long-continued, excessive dilatation of the cicatricial tissue; and he anticipated bronchoscopy by the invention of a tube which inserted into the larynx enabled the foreign body to be expelled.

From the pioneer work of these great contributors and those who have succeeded them, we have indisputable evidence that the fundamental principles of laryngology and rhinology as known today were born of the genius of our fellow countrymen.

Meanwhile, what has been the work of the American Laryngological Association.

WORK OF AMERICAN LARYNGOLOGICAL ASSOCIATION

The available evidence of the life and activities of the association is set forth in its published transactions. Since the inception of the association every year has been represented by a volume in which has been preserved the results of the best thought and effort of the foremost of our leading specialists, to the number of more than 1,700 original articles, Year after year these volumes have accumulated with absolute regularity, even during the most strenuous years of the war, although thirty-five of our members were absent at that time on active military duty. We may look with just pride on the record of their service as well as on these fifty volumes which so conclusively establish the position of our society as being the oldest and the most continuously active of its kind.

Reviewing the scientific material of our transactions, we find that in the study of the upper air passages the association has pursued a wide variety of subjects, intimately connected with its legitimate field. There has always been a strong desire to recognize the larynx as the point of all others deserving attention. Our statistics, however, show that while there have been presented to the association since its founding to date approximately 325 articles relating to the larynx, their relative proportion has declined with the advance of time. Thus, from 1879 to 1889, 103; from 1889 to 1899, 66; from 1899 to 1909, 76; from 1909 to 1919, 44, and from 1919 to 1928, 36. This should not be.

The annual addresses of former presidents of the association have offered much in the way of stimulation and suggestion to the student of today. Thus, Dr. Elsberg in his inaugural announced as the aim of the association "The promotion of knowledge in all that pertains to the diseases of the upper air passages," and suggested various activities in which

the association might well interest itself. At the outset he urged that the association should become the means of introducing those of kindred tastes in the practice of medicine and of fostering the friendships thereby made. The address of Dr. Rufus P. Lincoln, in 1900, was a masterpiece of thought and style. In his address in 1901, Dr. Swain urged the necessity for the strong personal interest of members in the welfare of the association—an interest which should be preeminent. Swain's advice as to the education of students in laryngology remains in force today and is well worthy of remembrance. Dr. Bryan, in his address in 1903 celebrating the twenty-fifth anniversary of the society, gave an interesting resume of its progress which to that date had included the furnishing of 548 scientific papers. The suggestion of Dr. Bryan that a monument be erected to Dr. Joseph O'Dwyer is still pertinent.

The fact that our transactions are not known to all our members is sometimes manifest. From the first of them to the last they are invariably interesting, whether studied to learn which of the ideas and suggestions have survived, which have been superseded, and which have been forgotten. No period could be more appropriate than this for a careful survey of their content—a task which could not fail to reveal much valuable information and throw light upon not a few forgotten and neglected treasures.

As a factor in the introduction of our science to this country the association has from the outset played the leading part. Its members have been the inspiring instructors in all of the centers of teaching. With such masters as Solis-Cohen and Allen and Sajous in Philadelphia, Oliver and Knight and Hooper in Boston, Elsberg, Wagner, Lefferts, Bosworth, in New York, French in Brooklyn, John Mackenzie in Baltimore, Shurly in Detroit, Ingals in Chicago, Glasgow in St. Louis, de Roaldes in New Orleans, Logan in Kansas City, and those who have followed, laryngology has reason to be proud of its sponsors.

In his presidential address in 1906, Gleitsmann comments on the progress of teaching at that time, as follows: "The position of laryngology in medical colleges and universities is more gratifying in the United States than abroad. Thus, in twenty-nine colleges of the United States having the largest number of students it was taught by fourteen professors in ordinary, in two by so-called special professors, in another two by professors residing elsewhere. Clinical professors taught in eight, lecturers in two, and in the remaining six there was no teacher of laryngology. Comparing conditions on the continent, it was found that B. Frankels' assertion was still true, namely, "We have not yet a regular professorship for laryngology. The majority of teachers in the twenty German and Austrian and the five Swiss colleges are so-called extraordinary professors, with the exception of Brussels and Rostock,

where laryngology combined with otology is taught by a full professor." Now, after twenty years, the advances made relatively here and abroad have not materially altered the situation. Every day is bringing new evidence, much of it from unprejudiced outside observers, of the rapidly growing superiority of our own special institutions and the character of the instruction which they afford.

The experience of fifty years has clearly demonstrated the value of broad investigation based on modern scientific lines. It is time that a new era in laryngology should be introduced through the employment of this method for the solving of our problems, especially those which relate to some of the most common and at the same time most pernicious ailments. Only within the past few months the study of the so-called "common cold" has been scientifically undertaken, although vast sums have been and are being expended on the attempted discovery of the causes and cure of cancer, tuberculosis and other diseases far less prevalent.

While the years have brought innumerable betterments in our knowledge of the diseases of the upper air passages, a never ending vista still leads to the things unknown. Thus, in many instances vasomotor coryza still defies us, questions relating to the diseases of the paranasal sinuses are unsettled, and many other conditions remain to be explored.

One of the most brilliant examples of the intelligent conservation and grouping of the details pertaining to the histories of large numbers of important cases is shown in the extensive collection made by Dr. Chevalier Jackson of foreign bodies extracted by him from the air passages. These, with their histories, constitute a complete compendium of instruction and impart a striking lesson.

Another member of the association, Dr. MacKenty, has done valuable service in the department of laryngectomy not only by reason of the number of his cases in which successful operation has been performed, but by the painstaking compilation of their histories, from which he has made possible deductions of actual scientific importance, in like manner with Sir St. Clair Thomson in the study of laryngofissure. These are promising beginnings for a series of investigations which shall review the experiences of the past, eliminate its faults, recognize its successes, and formulate methods embodying the practices which have been proved the best.

The age old discussion of the tonsils has become so trite as to make it almost the opprobrium of our medical gatherings. But with all the efforts put forth little precise knowledge has been established. A valuable step toward the better understanding of its pathologic changes has been made by the recent work of Dr. French. Such contributions as his should be received with the seriousness which their value demands.

Every one knows the benefit derived from the proper treatment of diseased tonsils. How much better would it be for the patient if the diseased tonsils were not allowed to develop. There is no more urgent need today than that of an intensive, thoroughly scientific investigation of the physiology and pathology of the lymphoid structures of the pharynx, especially as relating to causes, metabolic or otherwise, which contribute to its common pathologic conditions. I earnestly commend this to your early and serious attention.

More attention should be paid to the study of the physiology of the larynx, with especial reference to the production of the voice. Since the days of that greatest of all masters of tone production, Manuel Garcia, there have been other teachers of excellence and many fine singers have been developed; but even with the advanced instruction of the present day much improvement is desirable, while the quality and use of the voices of people in general is such as to call for our earnest attention, whether regarded from the point of view of esthetics or from the actual physical well being of the individual. One of the most important contributions made by our association to laryngology is the method of laryngeal photography originated and introduced by Dr. French more than forty years ago and now forgotten. Aided by this perfect method, Dr. French made elaborate studies not only of pathologic conditions but of the vocal bands in action. He explained the technic of the production of the various tones of the human voice, as demonstrated in the larynges of highly trained artists, both male and female, and was able to confirm or to correct the views empirically derived by the best masters of singing. That others should not have taken up the studies which he carried on with such scientific zeal and artistic demonstration has involved the loss of a great opportunity for scientific advancement.

GROWTH OF ASSOCIATION

The association has always maintained itself on a basis of financial independence, but there is no question that an endowment of moderate proportions might furnish the means of increasing its usefulness. Thus, in suitable cases research work might be encouraged; as, also, the publication of valuable scientific work—possibly too voluminous to be otherwise included in the transactions; the traveling expenses of members of the council in attending to the work of the association; provision for the entertainment of distinguished guests and specially invited lecturers; and such other matters as might legitimately appeal. Already a nucleus for such a fund has been provided through the generous foresight of Dr. Samuel Johnston.

It has long been my ambition that the association might have for itself a general rallying place, a home which might be an established

center for its activities other than its meetings, and be a depository for its material treasures. In it should be deposited the archives of the association, its memorabilia, the portraits of its members and of other distinguished colleagues, authoritative descriptions of original discoveries and new methods, and collections of objects of interest of all kinds pertaining to the history and development of our specialty. For this, there should be an efficient custodian, thoroughly qualified by nature and experience and with a long tenure of office. Such a scheme would serve many useful purposes, some of which are obvious. A highly important function might be created, hitherto untried, a collection representing the original models of instruments and methods, for the purpose of placing these on record and on visible exhibition, to the end that the claims of priority of the actual inventor might be secured to him, and that recurring repetitions of undesirable instruments might be prevented. Also in the presentation of original matter of any kind there should be some established and recognized authority for verifying the record for the settlement of questions pertaining to claims and for the proper adjudication of credit. The conclusions thus arrived at would be permanently recorded for the reference and guidance of any who in the future might be interested. Had this method been pursued in the case of the department of laryngology, the United States would long ago have secured the credit which it so richly deserves from the world at large.

The originality and scientific value of the work so auspiciously begun by our association, has been continued throughout its life with unabated energy. Time is lacking to recall all of the notable contributions of its members or to give due credit to the men themselves. Our Fellows have been leaders as discoverers, inventors, educators, writers and students. Such names as those who have gone before, and the splendid array of those now living who are "carrying on," fully sustaining the ideals and the advances of their predecessors, assures us of the continued preeminence of our country in the developments and achievements of laryngology and of our association as its worthy representative.

Let us, individually, continue to pursue the work which providence has placed in our hands, with abiding enthusiasm and a faith which "age cannot wither nor custom stale." Our association has offered more than one brilliant example of sustained professional interest and still provides them, as witness J. Solis-Cohen, Ingals, French, Pearce, Birkett, Bryan, Swain, Desverinne, Schmiegelow, and our distinguished guest. Such careers are splendid inspirations to those who are conscious that the years bring with them grave responsibility of utilizing the rich accumulation of their experiences. Work—the magic watchword of Pasteur, the stimulant of our energies, the solace of our trials, the promise of the fulfilment of our hopes, the only material thing that counts—while the days last.

And so, always growing in strength, in prosperity and in usefulness, with the close of our first half century we enter on a new era of endeavor, confident of the future because stimulated by the achievements of the past; with full faith that the same wisdom, foresight and unselfish devotion which created the American Laryngological Association and established for it the high ethical and scientific standards under which it has flourished preeminent for fifty years will be continued with unabated loyalty and zeal; and, so continued, will carry forward its mission of truth and light with ever increasing renown.

LARYNGEAL SENSORIMOTOR NEUROSIS

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Sensorimotor neuroses of the larynx are conditions frequently met with and often incorrectly diagnosed, especially by physicians who are not laryngologists.

My observation and study of these laryngeal manifestations led me to classify them under two heads, namely: laryngeal sensorimotor neurosis simplex and laryngeal sensorimotor neurosis grandé. Both conditions are neurotic and come under the broad classification of hysteria. The latter, however, may continue with uninterrupted symptoms for weeks and months or even for years. Neurosis simplex is frequently seen by laryngologists, while neurosis grandé is so rare that I was unable to find in the literature the history of a case such as I wish to present.

Simple laryngeal neurosis presents itself in various forms. There may or may not be a neurotic family history; however, careful investigation often presents some evidence of neuroses in the individual or in some of the progenitors. It is more common in women than in men and usually occurs between the ages from 15 to 35 years.

The chief symptom is aphonia, which may be partial or complete, and comes on suddenly without any apparent cause. Every conceivable type of loss of voice may be present which lasts from a few seconds to several days. Uncomplicated laryngeal neurosis simplex does not show any constitutional symptoms.

Examination of the larynx is important, but even this may sometimes be misleading. It must constantly be kept in mind that in this condition the larynx is always free from any evidence of inflammation, e. g., the entire mucous membrane is of normal pink, the vocal cords are pearly white and their margins smooth and cleancut. There is no evidence of infiltration or thickening about the arytenoids or false vocal cords; however, there may or may not be a partial or complete disturbance in adduction of one or both vocal cords. Such a condition of the cords adds greatly to the difficulty in diagnosis, as extralaryngeal conditions, such as pressure from tumors, etc., on the recurrent laryngeal nerves, or a neuritis involving these nerves, may cause bilateral or unilateral adductor paralysis while the larynx itself does not show any evidence of inflammation. The careful history and examination of the patient will generally enable one to make an accurate differential diag-

nosis in these conditions. Partial anesthesia of the larynx and often of the pharynx may be present in laryngeal neuroses, although hyperthesia may be part of the syndrome.

The important diagnostic points in laryngeal sensorimotor neurosis simplex are: Aphonia, sudden in onset, without constitutional symptoms to indicate the presence of some extralaryngeal disease which might affect the nerve supply of the larynx or of the laryngeal adnexia or any evidence of intralaryngeal inflammation.

As I stated at the outset, laryngeal sensorimotor neurosis grandé is evidently exceedingly rare, or cases have not been presented in the literature. It differs from the simple form only in that the symptoms may continue unbroken for months and as in the case I wish to present at this time, may last for years.

REPORT OF A CASE

Mrs. P., white, aged 32, a trained singer, had never been sick a day in her life, except for some of the diseases of childhood, and did not give a history of a neurotic tendency. She was asked to be soloist at a religious revival meeting. After singing with perfect ease the previous night, on the morning of Oct. 10, 1926, she suddenly partially lost her voice. This condition remained continuously until I saw her on May 26, 1928. She was a woman of splendid physique, clear skin and normal weight. She had never had any trouble with her throat during childhood, not even an attack of tonsillitis or slight irritation which might necessitate the occasional clearing of her throat, and she had never had a cough. She said that about one and one-half years ago she came to my office to consult me, and found me absent from the city. Since then she has been examined by a number of laryngologists in various cities and had been treated by several. I am inclined to believe that all overlooked the real factors leading to an accurate diagnosis of her case.

Physical examination revealed a normal nose and mouth with a complete upper and lower set of healthy, beautiful teeth. The pharynx was normally moist, smooth and free from any inflammation or granulation. On the advice of a prominent laryngologist to have her tonsils removed, Dr. C. I. Childers, her physician, removed her tonsils over one year ago. Dr. Childers insisted from the outset that he could not see any indication for the tonsillectomy, as the tonsils were small and did not show any evidence of infection. It is my opinion that noninfected tonsils should never be removed, more especially in singers as I believe they help to keep the throat moistened and hence render phonation easier. A definite indication, therefore, should be present in these cases for such an operation.

Mild silver protein, neo-silvol and chloretone were almost universally prescribed in the treatment by the various throat specialists and general physicians with as near as possible absolute rest to the voice. There was complete absence of any irritation or inflammation in the nasopharynx or about the posterior pharyngeal wall. The true vocal cords were pearly white and the edges cleancut. The epiglottis was normal, and there was no evidence of roughness or infiltration about the arythenoids, the interarythenoid notch or the false vocal cords. There was marked anesthesia both about the pharynx and larynx so that it was easy to make a careful and extended examination of the larynx by the indirect

method. The arytenoids and vocal cords acted in perfect uniformity as was excellently demonstrated to the assistant by having the patient laugh while watching the arytenoids and cords. The cords moved back and forth in perfect unison and harmony, nevertheless the patient could not phonate except in a hoarse low pitched voice or in a high stridulent tone. This condition had existed uninterruptedly for nearly two years in spite of all the strenuous treatment that she had undergone, a very serious condition for a singer.

The patient presented some interesting phenomena in phonation notwithstanding the apparently perfect action of both vocal cords.

When asked to do so, and while the larynx was being observed, she could pitch the voice to high A, but the tone would go quickly from a clear note to a split tone or to two distinct notes in the scale. This same was true when she was asked to make and sustain a low tone. This I know may indicate some impairment of the recurrent laryngeal nerve. Careful examination and observation, however, revealed positively that there was no impairment to the functions of either of these nerves. The results of all the laboratory observations were negative. With the evidence therefore of no intralaryngeal or extralaryngeal pathologic condition and the history of a sudden onset following a definite emotional disturbance, it was clearly a case of laryngeal sensorimotor neurosis, and in view of the fact that the condition had existed continuously for nearly two years, I called this condition laryngeal sensorimotor neurosis grandé.

The treatment of this patient, which had evidently been based on a diagnosis of chronic laryngitis, with rest to the larynx and medication with sedatives was entirely wrong. It was like putting a healthy elbow joint in a splint.

My treatment, which resulted in a complete cure so quickly that it seemed almost like a bit of magic, was as follows:

1. The patient was assured that she would get well in a very short time.

2. The larynx was to be used by running several octaves in the scale several times daily, keeping it up just to the point of not producing exhaustion, and increasing the amount of exercise daily.

3. Applications of drugs were not made to the larynx but (and I consider this most important) one-fourth grain (0.016 Gm.) phenobarbital tablets were prescribed, starting with two the first day and increasing the dose just to the point of producing slight drowsiness. In ten days, the patient's voice was as clear as ever only needing a little exercise to bring it to full strength of tone. I believe the phenobarbital should be discontinued in such cases as soon as possible, and if necessary, some other sedative may be used. The action of this drug on the cortex of the brain makes it of especial value in the treatment of such patients.

309 Colcord Building.

TELANGIECTASIS OF THE NASAL SEPTUM TREATED WITH RADIUM *

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NEW YORK

Telangiectasis, which is a varicose enlargement or dilatation of the veins or capillary vessels and minute arteries, is frequently encountered. The site is usually the anterior portion of the cartilaginous septum known as Kiesselbach's area.

This site is the junction of a branch of the superior maxillar artery, called the anterior artery of the septum, and the ascending branch of the palatine artery with numerous communicating branches. The mucous membrane over this area is thin; the arteries and veins are therefore superficial, and hence liable to the slightest injury. The veins are incapable of retracting into deeper tissues after being ruptured If any of these superficial vessels is defective it may give way with the slightest increase of blood tension.

REPORT OF CASES

CASE 1.—G. H., an unmarried girl, aged 17, a student, began to bleed first from one nostril and then from the other six years before she was admitted to the hospital. The family and past history were negative. She began to menstruate at the age of 12 and had been regular and normal. The bleeding was not profuse and was easily controlled. Two years before admission the nosebleeds increased in frequency and quantity, so that every two or three months she had a severe hemorrhage which was difficult to control. She had had treatment without effect.

Examination on June 12, 1925, showed the nose to be of the long, thin, sensitive type. The nasal septum was covered anteriorly with a network of dilated blood vessels which bled profusely when touched with an applicator. The septum was not deviated and no disease of the sinus existed. The tonsils had been removed successfully. An examination of the blood for bleeding time showed a normal reaction of three minutes and a normal coagulation time of four minutes.

A 10 mg. radium needle in a steel container was applied to each side of the nasal septum for two hours. Two such treatments were administered. There was no reaction.

Examination six months later revealed several blanched spots on the anterior part of the septum, and a network of narrow blood vessels which no longer bled when touched with an applicator. The result was gratifying, as the patient had not had nosebleed for an entire year.

The patient returned on June 19, 1926, stating that she had had two hemorrhages in the past week. Examination of the nose showed the posterior part of the septum normal in appearance and devoid of dilated blood vessels.

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* From the Oto-Laryngological Service of Beth Israel Hospital.

The anterior part of the septum for a space about three quarters of an inch in diameter was full of dilated and prominent blood vessels, on some of which small clots indicated where the last hemorrhage had occurred. One 25 mg. platinum radium needle without any screenage was applied and allowed to remain against the dilated blood vessels of the septum for two hours. There was a severe reaction which kept the patient uncomfortable for the entire week following the treatment. This treatment was repeated five weeks later. The reaction was not as marked as after the first treatment. Nosebleed did not occur between applications.

Examination three months after the treatments showed the entire anterior part of the nasal septum blanched with complete absence of any visible blood vessels. No further hemorrhage had occurred. Examination six months later revealed a similar condition. The patient was discharged as cured, but advised to return for observation every six months.

Case 2.—L. S., a man, aged 35, a printer, was referred to me by Dr. A. I. Ringer for relief of continuous nasal bleeding. A nasal operation, evidently a submucous resection, had been performed about four years before I saw him. From that time up to about a year before admission to the hospital he had had constant bleeding from the nose. The present condition dated back six months. At that time he had a severe hemorrhage from the left nostril. The bleeding was controlled only after a large amount of blood had been lost. After this the nosebleeds increased in frequency so that at the time of admission the patient was weak and anemic. The last hemorrhage occurred Jan. 12, 1927. Examination January 17 showed the man to be anemic, and the nasal septum showed a large septal perforation at the anterior quadrant. There was some oozing from the posterior margin of the perforation, the septum showing an area about 4 mm. in length covered with numerous small blood vessels which bled on touch. A number of dilated and tortuous vessels were present at the anterior parts of both sides of the septum. The inferior turbinae bone at the right side appeared to be exceedingly vascular at its anterior inferior margin, and several petechial areas were plainly visible. No sinusitis or other pathologic condition was present. It was interesting to note in this case the presence of numerous small round and indurated petechial areas on the tongue. An examination of the blood showed a normal reaction of four and a half minutes for bleeding time and five minutes for coagulation.

The treatment consisted of three applications of 25 mg. of radium element in the form of a needle screened with platinum and applied to each side of the nasal septum for three hours. These treatments were given on January 21, February 17 and March 10. No reaction followed. Bleeding ceased after the first treatment.

Examination three months after the last treatment revealed the margin of the septal perforation healed. The septum appeared blanched and all the dilated vessels had practically disappeared. Examination six months later revealed a similar condition; the patient had not had nosebleed since the treatments.

Case 3.—In Mrs. A. C., a housewife, aged 40, whose family and previous history were negative, the present illness dated back to two years before admission to the hospital when without warning or cause she had a severe nosebleed from the right nostril. This was followed by repeated nosebleeds from one or the other nostril, usually not alarming in severity. About two days before examination, she had a severe hemorrhage from the left nostril which necessitated repeated nasal packing before it stopped.

Examination on April 1, 1926, showed a slightly flattened nose with a marked deflection of the septum. There was a large spur on the left side which almost touched the base of the nose. There was a distinct ulcer present at the anterior part of the left side of the septum, which was undoubtedly the site of the nosebleed. The anterior parts of both sides of the septum showed a network of numerous blood vessels, some standing out distinctly, elevated and bleeding on touch. Where the spur on the left side of the septum took a sharp turn downward to the base of the nose the mucous membrane was extremely vascular and was probably the site of several previous nosebleeds. A chronic maxillary sinusitis was also present. This was confirmed by a roentgenogram.

The treatment consisted of two applications of 25 mg. radium element placed in platinum needles against each side of the nasal septum for four hours. A slight reaction followed, and the patient complained of a little pain on the tenth day. This, however, was not severe and lasted only one week. Bleeding did not occur after the first application of radium.

Examination of the nasal septum after three six month intervals revealed a blanched nasal mucous membrane with all its vessels obliterated.

This patient is now being treated for sinusitis and has not bled even after the application of suction to the nares up to ten points of pressure on the Sorenson gage.

COMMENT

In the first patient, the early treatment was not successful because of the small dose, and also because of too much screenage. With the increased dose and lessened screenage the bleeding ceased promptly. In the other two cases, cessation of hemorrhage was prompt and certain after the first application of the radium.

44 West Seventy-Fourth Street.

THE INFLUENCE OF FLUORINE ON THE BONY LABYRINTH OF THE WHITE MOUSE (MUS MUSCULUS ALBINUS)

PRELIMINARY REPORT *

ALFRED LEWY, M.D.
CHICAGO

Modern biochemistry and pharmacology do not show many studies of fluorine preparations. Its clinical application has been largely theoretical or empiric. Some of the studies are briefly surveyed here. The earliest reference found was the discovery of fluorine in fossil ivory by Marischini, 1803, quoted by Gmelin and Noger.[1]

Tappeiner and Obolonski [2] described its toxic effects as depression of the nervous system, fibrillary twitchings, convulsions, weakness, salivation and breathing hastened and deepened.

Brissemoret [2a] recommended calcium fluoride for introducing calcium into the body. He says it is as good as the carbonate or phosphate. He uses it for infants and adults being treated for caries or ununited fractures, and during pregnancy and nursing.

Zdarek [3] described the distribution of fluorine in the various tissues as found by him in man.

Haeger,[4] experimenting on rabbits with the artificial increase in growth of bone for therapeutic purposes, found the greatest increase from plugs of calcium fluoride placed in holes bored under the epiphyses as compared with the effect of similar plugs of calcium carbonate, magnesium phosphate and calcium phosphate.

Gautier [5] found fluorine in all tissues in this order: (1) those of low vitality; epidermal scales, enamel, hair and feathers; (2) bone, cartilage, teeth and tendons; (3) glands, nerves and muscles. He reported a fairly constant proportion to phosphorus, e. g., 1:450 in tissues of high metabolic activity, and 1:3.48 in human epidermis.

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* Read at meeting of the American Laryngological, Rhinological and Otological Society, May 3, 1928.

1. Trebitsch, Fritz: Biochem. Ztschr. **191**:237, 1927.

2. Tappeiner and Obolonski: Arch. f. exper. Path. u. Pharmakol. **25**:203, 1889.

2a. Brissemoret, A.: Bull. Gen. Therap. **156**:147, 1909.

3. Zdarek, A.: Physiol. Chem. **69**:127, 1910.

4. Haeger, F.: Inaug. Dissert., Greifswald, 1912.

5. Gautier, A.: Compt. rend. Soc. de biol. **74**:110, 1914.

In tissues of low metabolic activity, used for mechanical protection, the proportion is about that of the mineral fluorophosphates (apatite).

Schwyzer [6] found that sodium fluoride given intramuscularly caused loss of calcium and chlorine in bones, probably owing to increased resorption activity of bone cells. He found fluorine preparations poisonous in less than 1 mg. per kilogram.

Sontag [7] found fluorine in the bones and teeth of dogs raised by sodium fluorine feeding.

Wieland and Kurtzahn [8] concluded that sodium fluoride liberates fluorine ions which combine with calcium in the tissues, causing toxic effects. Hydrofluoric acid is caustic because of fluorine molecules, not because of acid reaction.

Sollman, Schettler and Wetzel [9] find 0.5 mg. per kilogram a fatal dose for albino rats. Smaller doses retarded growth, which was not altogether due to lessened appetite and intake. Growth remained permanently impaired in spite of withdrawal of fluorine. Calcium and sodium fluoride were used.

McCollum, Simmonds and Becker,[10] adding sodium fluoride 229 parts per million to the food of white rats, reported gross changes in the teeth and some changes in the bones of the skull. Schulz and Lamb [11] confirmed these observations.

CLINICAL APPLICATIONS

The earliest reference to the clinical application of calcium fluoride appears in the writings of Schuessler,[12] who founded what he called biochemic therapy. In the twenty-fifth edition of his book, he recommends the administration of calcium fluoride in small doses so as not to disturb the healthy cells, in conditions characterized by (1) a hard lumpy exudate on the surface of bone; (2) a relaxation of elastic fibers, hence an enlargement of the vessels, and (3) keratin exudates from the epidermis, forming crusts, and causing chaps and tears. He says in part, without quoting experimental evidence, that the fluorine is split off and combines with nascent hydrogen in the tissues to form hydrofluoric acid, which dissolves the exudate molecule by molecule.

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6. Schwyzer, F.: Biochem. Ztschr. **60**:32, 1914.

7. Sonntag, G.: Arbeiten Kaiserliches Gesundheits Amt, Berlin **50**:307, 1917.

8. Wieland, H., and Kurtzahn, G.: Physiological Action of Fluorine, Arch. f. exper. Path. u. Pharmakol. **97**:489, 1923.

9. Sollman, T.; Schettler, O. H., and Wetzel, N. C.: Pharmacol. **17**:197, 1921.

10. McCollum, Simmonds and Becker: J. Biol. Chem. **67**:553, 1925.

11. Schulz and Lamb: Science **61**:93, 1925.

12. Schuessler, W. L.: Biochemic Therapy, trans. from twenty-fifth edition. Philadelphia, Boericke and Tafel, 1916.

He is referred to principally in the homeopathic literature,[13] in which I find mention of its use in conditions of the ear, namely, calcareous deposits on the membrana tympani, sclerosis of the ossicles and petrosa; chronic suppuration and resorption of osseous growths on the ribs and on the tarsal and carpal bones.[14] Otosclerosis as a definite clinical entity does not appear to be recognized.

A more modern study of the effects of fluorine on animals and man was made by Prof. Hugo Schulz, director of pharmacology, University of Greifswald, who reviewed its former use in medicine and its use in industry as a preservative.[15] He recommended the use of calcium fluoride in doses of 1/100 and 1/1,000 grain for patients with chronic diseases of the bone and with disturbances of the teeth, and said that further investigation is warranted as this substance is probably very useful in a restricted field.

Escat[16] recommended fluorine in various forms, including the calcium salt, in the treatment of patients with otospongiosis, but merely as an adjuvant remedy. He prefers pituitary obtained from the anterior lobe, 1½ grains (0.096 Gm.), administered from two to four times a day for from fifteen to twenty days, alternated with phosphorus for a year.

THE PRESENT EXPERIMENT

In 1922, I began empirically the use of calcium fluoride, 8 grains (0.512 Gm.), of the 1:1,000 trituration in lactose daily, for otosclerosis. Believing that I had some encouraging results, and at the suggestion of Dr. Norval Pierce, to whom I communicated them, I began feeding experiments on white mice in 1925.

Twelve unpedigreed white mice, born and raised in the same establishment and under the same conditions, were used. Six were controls, six were fed calcium fluoride finely triturated (eight hours) with lactose in a proportion of 1:1,000, mixed with their food, which they took readily. At the start, all were young of adult size. The feeding period covered from six to nine months, at which time all the experimentally fed mice were dead; the controls were still alive, except one that was killed by accident. The experimentally fed mice gave evidence of general toxemia, lack of energy, roughness and irregularity of fur and easy tendency to collapse when handled for examination of the teeth.

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13. Boericke, W.: Materia Medica. Clarke: Dictionary of Medicine, Philadelphia, Boericke & Tafel, 1918.

14. Bell, J. H.: New England M. J., 1874, p. 320.

15. Schulz, Hugo: Vorlesungen über wirkung und anwendung der unorganischen Arzneistoffe, Leipzig, George Thieme, 1920.

16. Escat, E.: Monographie, Otolaryng. Internationale **7**:653, 1922.

The teeth did not show marked changes, as I had been led to expect, only a little discoloration. To the experimentally fed mice only one litter of three was born, all of which died in a few hours and were eaten by others.

In five of the specimens of experimentally fed mice, the stapes region was shown in the slides that were made, in one part of the cochlea, for comparison with the same region in controls. The experimentally fed mice, numbers 1, 2, 4 and 5, by comparison showed more bluish staining of the bone; the lacunae were larger and more abundant

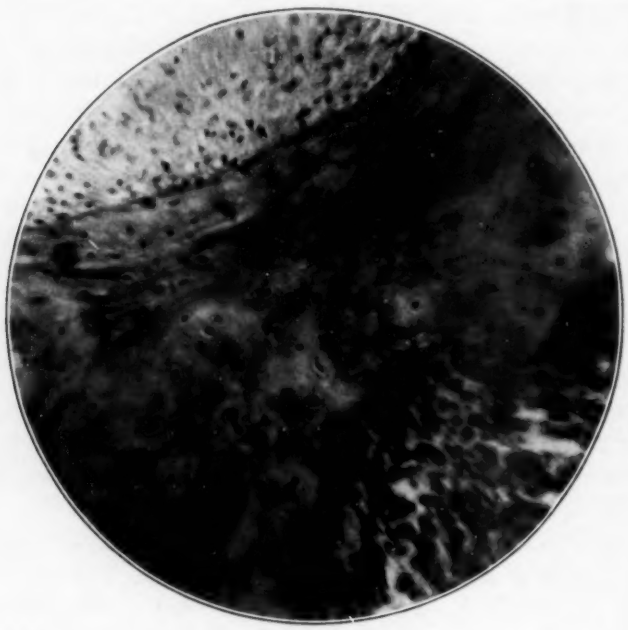

Fig. 1 (mouse 8).—Detail from portion of cochlea of control mouse; × 300. These photomicrographs are by R. D. Bensley.

and more regularly filled with bone corpuscles, which were also larger and more deeply stained; i. e., the appearance was that of calcified cartilage or of young or actively growing bone in adult mice, as if stimulation or irritation had occurred or perhaps merely inhibition or arrest of the maturing process. Experimental mice 3 and 6 showed no change. All these slides were run through by the same technician with the same technic (hematoxylin eosin stain), except one which was cut and stained by myself. These were all paraffin sections. Other sections from control mice now under preparation are being run through celloidin. So far only mature bone has been seen.

That fluorine must be reckoned with as a factor in bone metabolism has been shown by the investigators quoted. I believe that I have shown at least the strong probability that this influence extends to the bony labyrinth of the white mouse. To the otologist there naturally occurs the idea that with the proper preparation and application of fluorine one may be able to influence favorably the changes occurring in otosclerosis. This is of course a long step. In a disease subject to spontaneous fluctuations and arrest, and especially in view of the few sufficiently early cases seen by one man and the difficulty of holding

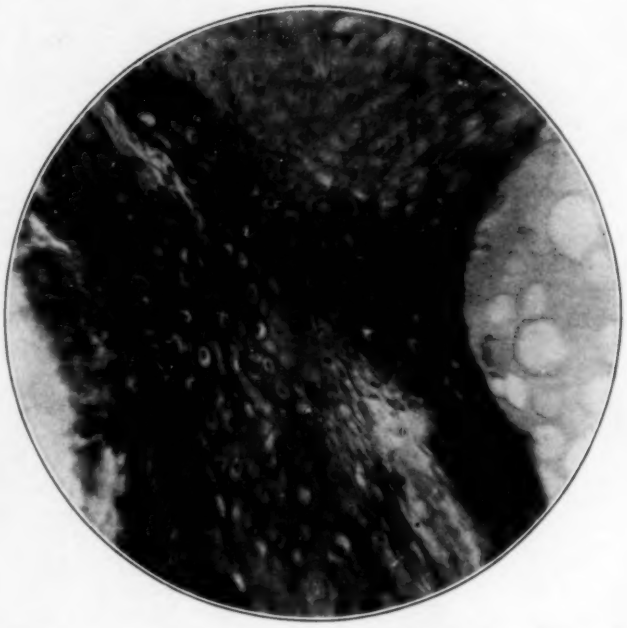

Fig. 2.—Detail from cochlea (mouse 4, fluorine fed). Note size and frequency of lacunae; × 300.

patients long enough for thorough study without resorting to so-called adjuvant treatment and psychologic methods that might in themselves invalidate the results, my results thus far are necessarily inconclusive, but not without some encouragement. A number of patients have progressed in spite of treatment, but slowly. Some have discontinued treatment, one in spite of apparent improvement; the others, it may be assumed, because of failure of improvement. On the other hand, a few appear improved even after several years.

My treatment consisted of 2 grains (0.12 Gm.) of calcium fluoride, 1:1,000, triturated eight hours with lactose, given three times a day on alternate months. No local or other treatment was used.

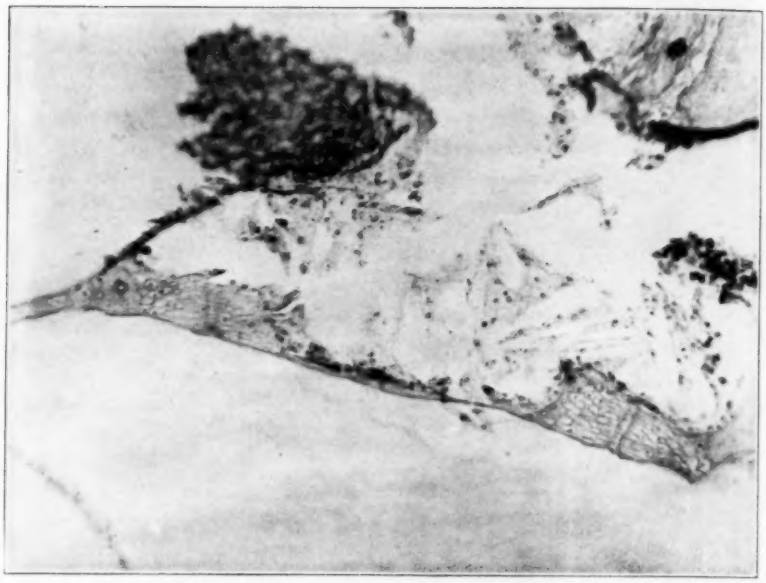

Fig. 3 (mouse 12).—Stapes region of normal mouse. The margin of the oval window and rim of the footplate of the stapes show many lacunae, mostly empty. The rest of the labyrinthine capsule is dense bone.

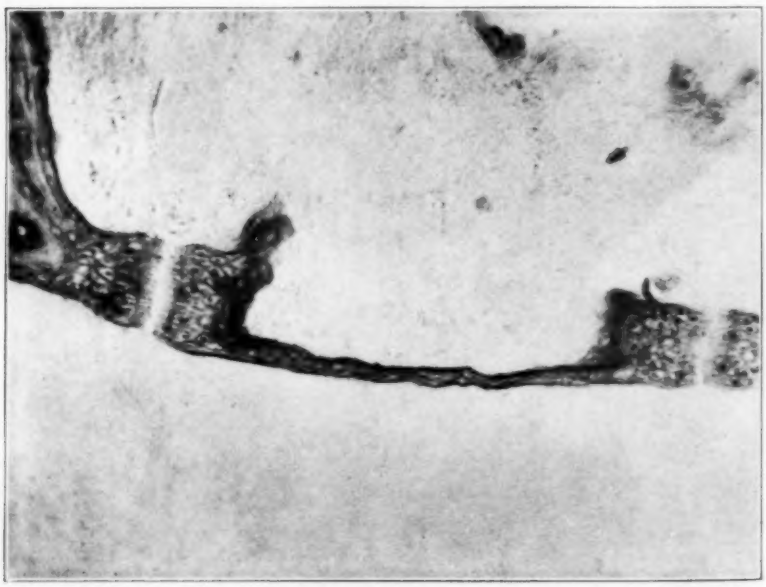

Fig. 4 (mouse 5).—Stapes region (fluorine fed). The lacunae are more regularly filled with corpuscles, and are seen to extend further into the capsule. The contrast with figure 3 is much greater in the stained slides than in the black and white picture.

REPORT OF CASES

CASE 1.—A. C., a woman, aged 30, whose family history did not show deafness, had been hard of hearing for six years, and had been compelled to give up her profession as school teacher. Paracusis willisii was present. The tympanic membrane was normal.

In March, 1925, the patient again began to teach, and was still doing so in May, 1928. Her family and friends insisted that her hearing was much better. On the audiometer record, she showed a 5 point increase all along the scale, but I was unable to demonstrate by the whisper test any definite and outstanding improvement. However, her limit of hearing for the lower tones was improved so that she could hear *A* (55 double vibrations), whereas before, the limit had

TABLE 1.—*Results of Tests Made in Case 1 on Aug. 21, 1924*

Tests	Right Ear	Left Ear
Whisper	High tones, 5 feet Low tones, 2 inches	High tones, 3 feet Low tones, 3 inches
Weber, not lateralized		
Schwabach, 75 seconds (normal 30 seconds)		
Rinne	— 48;17	— 48;17 (normal + 12;60)
Low limit	F # (90 d. v.)	F #
High limit	e4 shortened	e4 shortened
No improvement on inflation		

TABLE 2.—*Results of Tests Made in Case 2 on Aug. 2, 1927*

Tests	Right Ear	Left Ear
Whisper	22 feet	Low at 4 feet, high 6 feet
Weber	.....	Not lateralized
Schwabach	.....	45 seconds (normal 30)
Rinne	+ 28;30 (normal+12;60)	— 28;18
Low limit	A1 (55 d. v.)	F # (90 d. v.)
High limit	e4 normal	e4 shortened
Membrana tympani	Pinkish	No light reflex
Eustachian tube	Open	Open
No improvement after inflation		

been *F#* (90 double vibrations). I am convinced that there are some factors in hearing that the present tests do not reveal. They may be psychologic, but they are real.

CASE 2.—This was the only fairly early one of my cases. The mother of the patient was deaf, giving the tuning fork reaction of nerve deafness. B. S., aged 18, the patient, had noticed some difficulty in hearing for three years. Endocarditis had followed rheumatism in 1917; tonsillectomy had been performed in 1920. Hypersthetic rhinitis had been present in 1927. At the time of this report the patient's condition was better.

On Dec. 2, 1927, the low limit of hearing for the right ear was *D1* (36 double vibrations), for the left ear *A1* (55 double vibrations); on March 17, 1928, the low limit for the right ear was *B2#* (30.5 double vibrations); for the left ear *A1* (55 double vibrations). A whisper was heard by the right ear at a distance of 22 feet when the face was turned away from the speaker; low tones were heard by the left ear at a distance of 2 feet; medium tones at a distance of 18 feet.

Case 3.—N. A., aged 21, had been hard of hearing for two years; two brothers had been deaf since they had been in army service, 1918.

While presenting symptoms of conduction deafness of severe grade without evidence of inflammatory changes, the patient was practically totally deaf on the right ear. She heard the *a1* fork slightly, but with the noise apparatus in the left ear, nothing was heard. This is not a very positive case of otosclerosis. In June, she heard definitely on the right side when the noise apparatus was in the left ear, and the distance at which some words spoken in a conversational tone could be heard had increased from 12 to 16 feet. She herself claimed to hear "much better."

Case 4.—D. S. began treatment in 1922. My diagnosis was early otosclerosis, confirmed by Dr. Shambaugh. Because of the improvement I doubted the diagnosis at that time. She was not in my care for many months, during which time

Table 3.—*Results of Tests Made in Case 3 on Jan. 21, 1927*

Tests	Right Ear	Left Ear
Conversational tone	Totally deaf with noise apparatus	12 feet
Whisper	.....	6 to 12 inches
Schwabach	.....	45 seconds
Weber	.....	Not lateralized
Rinne	+ 20;15	+ 20;25
Low limit	.....	D 1
High limit	c4 short	c4 short
Membrana tympani	Delicate; red reflex	Red reflex
Eustachian tube	Open	Open

Table 4.—*Results of Tests in Case 4 Made Aug. 4, 1922*

Tests	Right Ear	Left Ear
Whisper	8 feet	3 feet
Schwabach	.....	60 sec.
Rinne	+25:40	—18:25
Low limit	55 d. v.	55 d. v.
High limit	c4 slightly short	c4 short
Eustachian tube	Open	Open

she took a large amount of acetylsalicylic acid for headache, due to some pelvic trouble which has since been corrected. When she returned to me in August, 1925, hearing had failed definitely. She was intermittently under treatment until January, 1928 (she lives in another state). The whisper test shows a further slight decline in hearing, but the low limit of hearing is unchanged.

After inflation of the right ear, a whisper could be heard at a distance of 12 feet; the left ear did not show any improvement.

The results of a subsequent test made on April 24, 1923, are as follows:

Before inflation, a whisper was heard by the right ear at a distance of 16 feet; after inflation, at a distance of 20 feet; the low limit was 36 double vibrations. The left ear could hear low tones at a distance of 3 feet, and medium tones at 5 feet.

The results of tests made on April 21, 1928, are as follows: A whisper could be heard by the right ear at a distance of from 8 to 18 feet; there was no improvement on inflation. The left ear could hear low tones at a distance of 10 inches, and other tones were heard at 2 feet.

CASE 5.—The father of Mrs. E. A. H., aged 37, was hard of hearing when he reached middle age. All paternal uncles and two aunts were deaf. One sister somewhat deaf; she died at the age of 21. The patient had received some treatment during adolescence for the condition of her ears.

On Nov. 11, 1925, a whisper was heard by the right ear at a distance of 22 feet plus. Low tones were heard by the left ear at a distance of 1 foot; high tones, at 6 feet. The low limit of hearing for the left ear was 55 double vibrations. Treatment was interrupted until April, 1928; the audiometer then showed a slight percentage of loss of hearing since Nov. 11, 1925.

TABLE 5.—*Results of Tests Made in Case 5 on March 27, 1925*

Tests	Right Ear	Left Ear
Whisper	16 feet	Low tones 6 in., high 5 ft.
Weber	.....	Lateralized left
Schwabach	.....	50 seconds (normal 30)
Rinne	+ 33;40	— 33;15
Low limit	36 d. v.	90 d. v.
High limit	c7	n6 (monochord)

TABLE 6.—*Results of Tests Made in Case 5 in 1925 and in 1928*

	C	c	c1	c2	c3	c4	c5	c6
Right ear								
11/11/25	25	11	14	12	11	0	0	0
4/12/28	33	22	18	16	11	12	13	16
Left ear								
11/11/25	49	39	27	40	27	23	17	38
4/12/28	57	51	36	40	23	23	26	32

TABLE 7.—*Results of Tests Made in Case 7*

	C	c	c1	c2	c3	c4	c5	c6
Right ear								
11/20/25	57	50	40	48	54	52	56	?
4/13/28	57	40	35	44	50	47	50	?
Left ear								
11/20/25	17	14	18	24	27	27	30	27
4/13/28	17	12	14	22	23	27	26	16

CASE 6.—Mrs. V. A., aged 38, suffered from severe conduction deafness. A conversational tone was heard at a distance of 6 inches by the right ear; at 2 inches by the left ear; a whisper was heard only when the lips were placed next to the ear. She could not hear a voice over the telephone, although according to the Schwabach test, the time during which the tuning forks was heard was greatly prolonged. After five months, the patient said that she could hear over the telephone. Tests did not show any change in her ability to hear. She has discontinued treatment.

CASE 7.—A physician whose case was diagnosed by Dr. Norval Pierce as otosclerosis had treated himself under my direction. An audiometer record of the percentage of loss of hearing showed slight improvement, seen in table 6.

Case 8.—Mrs. N., aged 40, came to me in 1922 with acute suppurative otitis media of the right ear. Her father, paternal aunt and two cousins on the paternal side, were deaf and had paracusis willisii. The mother had been deaf since childhood, following typhoid. After recovery and after inflation no longer brought about improvement, the patient was still very deaf and had paracusis willisii. My diagnosis was advanced otosclerosis. An early record was not available. There was apparent improvement for two years. Comparison of an audiometer record for 1925 and a recent record show a small percentage of loss in hearing.

COMMENT

These few cases are not conclusive from a clinical standpoint, but they have changed my attitude. I now feel justified in taking a hopeful attitude toward the patient with otosclerosis where I formerly had a discouraging one. In the future I shall not rely on fluorine alone, as was done in these cases for test purposes, but shall combine this medication with other preparations, such as parathyroid, pituitary obtained from the anterior lobe, or phosphorus, and such other measures as may be indicated by the general condition of the patient.

Table 8.—*Results of Tests of Hearing Made in Case 8 in 1925 and in 1928*

	C	c	c1	c2	c3	c4	c5	c6
Right ear								
10/20/25.....	74	51	41	36	29	22	10	32
4/13/28.....	74	51	45	40	32	24	26	27
Left ear								
10/20/25.....	?	67	54	54	48	41	38	?
4/13/28.....	?	73	63	56	48	47	38	?

Proper pursuit of this subject should be made by the biochemist and histologist. A mere practicing otolaryngologist like myself may follow the clinical course, but one man sees relatively few cases, especially few early ones. It is therefore suggested that the American Laryngological, Rhinological and Otological Society may see fit to further this work through more adequate channels.

CONCLUSIONS

Fluorine is a factor in bone metabolism with which one should reckon. In the white mouse, its influence appears to extend to the bony labyrinth of the ear.

To the otologist this suggests the possibility of its application in otosclerosis, and this subject is worthy of the attention of persons and of institutions properly equipped to do the necessary research.

It is hoped that the American Laryngological, Rhinological and Otological Society will see fit to further this end.

Drs. Roy M. Grinker and Georgianna Theobald gave valuable assistance in the preparation of specimens. Slides were also examined by H. Gideon Wells, pro-

fessor of pathology, Esmond R. Long, associate professor of pathology, Dallas; B. Phemister, professor of surgery, University of Chicago, and R. Myron Strong, professor of anatomy, Loyola University Medical School. The three first mentioned state that there are definite deviations from normal adult bone, but that they cannot classify these changes with anything with which they are familiar. Professor Strong, who has studied ossification in rats, says that the changes found most nearly resemble the condition found at the epiphyseal ends of the bonds of rats where ossification is long delayed.

25 East Washington Street.

ABSCESS OF THE LUNG FOLLOWING TONSILLECTOMY

REPORT OF A CASE *

RICHMOND McKINNEY, M.D.
MEMPHIS, TENN.

Lois M., aged 15, was operated on by a general practitioner on Oct. 1, 1927, for removal of her faucial tonsils. She apparently recovered from the operation, and returned to school, but about two weeks later a pain developed in the upper portion of the right side of her chest, with severe headaches and nausea, confining her to bed. The physician who performed the operation was called, and gave his opinion that she had pyelitis and sinusitis. She had fever at the time, the temperature running to 102.5 F., but never higher. She expectorated considerable blood and mucus from time to time. Infection of the sinuses was not revealed by the x-ray. A genito-urinary surgeon was consulted, and he advised that the patient be removed to a hospital for cystoscopy. She remained in the hospital about a week. Her temperature was said to have returned to normal, but on her return home, according to her parents, she began expectorating dark, foul-smelling pus.

While the patient was in the hospital, there was an epidemic of Vincent's angina among the nurses; the patient was infected, and developed the disease ten days after her return home. For this she was given arsphenamine intravenously. Subsequently, she lay in a stupor, and her kidneys ceased functioning. She was then taken to another hospital, and cystoscopy was again performed. While she was there, x-ray pictures were made of her chest, and an area apparently of abscess formation was discovered (fig. 1). An internist was called. He pronounced her condition abscess of the lung, and advised that she be referred to me for bronchoscopic treatment. About two weeks later the patient was seen by me at the Memphis Eye, Ear, Nose and Throat Hospital. She presented a most distressing picture; was greatly emaciated, and had a deep cough, with frequent expectoration of a large amount of foul-smelling, thick mucus. Her temperature, I was informed by her parents, had been around 102 F., without remission, for some time. There was only a moderate leukocytosis, 9,200, with 75 per cent polymorphonuclears. Tubercle bacilli were not found in the sputum.

Bronchoscopy was performed immediately, and the tube passed readily into an abscess cavity in the upper lobe of the right lung, involving at least a third of the lobe. When this was reached the odor was so foul that some of the spectators left the room. In the various patients with abscess of the lung whom I have treated, I have never encountered one with a case so disagreeable as this. The pus did not come through the tube on account of its thickness, but when it was diluted with warm saline solution, I was enabled to remove about 4 drachms of pus by suction. Two days later, I again performed a bronchoscopy, and about the same amount of pus was suctioned out. Three days later, the procedure was repeated. On January 11, just one week subsequent to the first bronchoscopy, a transfusion was given. It was practically impossible to get a needle into the median basilic vein of the left arm, owing to the fact that several intravenous injections of arsphenamine had previously been given in this vein, and it had

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* Read before the American Bronchoscopic Society, April 30, 1928, Washington, D. C.

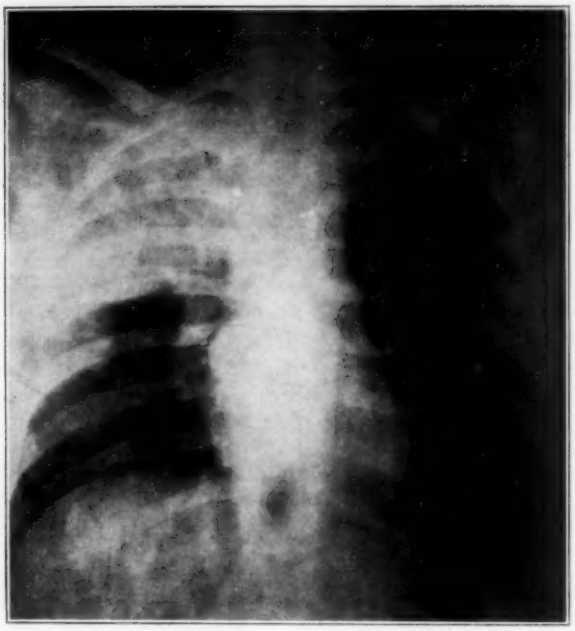

Fig. 1.—Abscess in right upper lobe.

Fig. 2.—Pus has drained from abscess cavity, and extensive pleurisy is shown.

collapsed. I cut down to the vein and succeeded, finally, in getting about 175 cc. of citrated blood into the circulation. A marked improvement in the child's condition resulted, her temperature going down to below 101 F. for the first time since she had been in the hospital, becoming normal several times and ranging around 100 F. On January 14, bronchoscopy and suction were again performed. On January 18, in the afternoon, there was a marked change for the worse, the temperature suddenly rising to 103 F., following a rigor, with rapid remission. The temperature continued to fluctuate in this manner for several days, but never again went so high as 103 F. On January 24, another transfusion was given, the right arm being used. Difficulty was not encountered in making entrance into the median basilic vein, 500 cc. of citrated blood being given. The condition did not improve following the transfusion. Another x-ray picture was made; it showed the abscess cavity clear of pus, but there was an extensive pleurisy (fig. 2). The patient was complaining of severe pain in the chest, and the expectoration of foul-smelling pus had increased. At a consultation as to whether the lung should be collapsed or drained by resection of a rib, my advice was that neither measure should be taken, as I was convinced that the child was going to die soon, and I did not approve of subjecting her to unnecessary and useless discomfort. The next day, however, the patient was removed to another hospital, a rib was resected under local anesthesia, and a large quantity of pus was drained out. She died two and one-half hours later.

It will be noted from the x-ray pictures that the area of the upper lobe which originally showed cavitation and pus appears clear in the final picture, although an extensive pleurisy is seen. Perhaps this was due to erosion and discharge of the pus to the pleura, resulting in pleurisy, which was the immediate cause of the child's death.

I cannot help feeling that the patient might have lived if bronchoscopic treatment had been administered early. All indications pointed to the presence of an abscess of the lung two weeks subsequent to the operation, and bronchoscopic treatment administered at that or shortly thereafter would, I believe, have resulted in the saving of this patient's life.

TUMOR OF THE HYPOPHYSIS

REPORT OF A CASE *

F. H. LEE, M.D.
RICHMOND, VA.

This case is reported because of its interest to the rhinologist. The only objective symptom the case presented, other than the x-ray picture, falls in the field of rhinology.

REPORT OF CASE

History.—M. E. J., a white man, aged 57, was seen on Feb. 2, 1928, complaining of almost constant frontal and left temporal headache of five months' duration. The pain extended to the ears as a sensation of discomfort and of stuffiness. An area of soreness was present in the left posterior temporofrontal region from which there was an occasional shooting pain to the left ear. The headache was usually relieved with acetylsalicylic acid for from six to twelve hours. During the earlier part of this period the exacerbations of pain were of greater severity than they were during the latter part. For two months, the hearing had been diminished in the left ear; the hearing in the right ear had not been good since childhood, when the patient had suffered with abscesses of both middle ears. Usually there had been a small amount of sticky postnasal discharge in the mornings.

Before the onset of this trouble, the patient had been healthy. Some years ago he had been given x-ray treatment for an enlarged cervical gland and had been told that the condition was Hodgkin's disease, but later the roentgenologist had denied this, saying the condition was one of simple adenitis.

Treatment of his present condition by several physicians had not been of any avail. Dr. A. A. Houser, whom he had last consulted, referred him to me with a report of a careful general examination, with negative results, including complete laboratory work. Dr. Houser expressed the belief that the patient was suffering from an intracranial condition in spite of the fact that a roentgenogram had been made and reported negative for tumor of the brain.

Physical Examination.—On examination of the nose, neither obstruction nor secretion was found. The mucous membrane was not congested, hypertrophied or atrophic. Tenderness of the sinuses was not present, and light transilluminated the antrums and the frontal sinuses equally and well. There was a small amount of sticky, grayish mucus in the nasopharynx. The results of examination of the throat were negative.

Inspection of the ears revealed retracted and partially atrophic drum membranes. The hearing was diminished. The watch was heard at a distance of one-half inch, and all forks were heard but diminished in time. Bone conduction was greater than air conduction.

Examination of the eyes revealed the following: The appendages and anterior segments of the eyes were normal, with the exception of the left pupil, which was slightly irregular. The refractive media were clear, and the fundi were normal. The retinal veins were a little full. There was no muscle imbalance. The form

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* Read before the Richmond Ophthalmological and Otolaryngological Society, May 14, 1928.

fields were normal. There was no enlargement of the blind spot. The tension was 35 degrees in each eye with a McLean tonometer. Vision in the right eye was 20/100, and was not improved with his glasses; that in the left eye was 20/100, and was corrected to 20/30 with his glasses. The patient was wearing a plus 1.50 spheric lens before the right eye and a plus 3 spheric lens before the left eye. On manifest refraction, the patient accepted a minus 1 with a plus 2 axis 105 for the right eye with resulting visual acuity of 20/30. The refraction with retinoscopy under homatropine was minus 1 in the right eye with plus 2 axis 105, and plus 2 in the left eye with plus 0.25 axis 180, with a visual acuity of 20/30 and 20/20, respectively. The retinoscopic refraction with a plus 2.5 added for near vision was given. It may be noted here that no part of the ocular syndrome of pitutiary tumor was present.

Lateral view of the head showing erosion through the floor of the pituitary fossa.

Treatment and Course.—On February 6, four days after having been given glasses, the patient returned but had not experienced any relief from his complaint. The sphenoid sinus was then washed on the left side after easy cocainization of its anterior wall. A small frontal sinus irrigator, no. 5 French, was used.

With the probe, the posterior wall gave the sensation of being covered with granulation tissue instead of the hard, firm, bony wall that is usually encountered. On withdrawing the probe, the nose bled slightly from above and behind the middle turbinate. The sinus was then washed with a slightly blood colored but otherwise clear return. When the posterior wall of the sinus was touched with the probe, it caused pain which radiated to the sore spot on top of the head and

which the patient thought was unmistakably connected with his complaint. At this time, I felt that the patient had an infection of the sinus, probably of the streptococcic type.

On February 8, 10, 14, 16, 18 and 20 the patient was seen, and irrigation of the sinus and simple sprays were used alternately. The ears were inflated twice during this time, and inspection of the posterior nares with the nasopharyngoscope was made with negative observations, with the exception of redness of the mucosa covering the under surface of the sphenoid. The patient was easy to handle, cooperative and was sincere when he said at the end of this time that he had experienced absolutely no improvement from anything that had been done.

I then advised that relief might be secured from better drainage and ventilation of the sphenoid sinus by removal of its anterior wall, but requested roentgenograms of the sinus before resorting to this procedure.

X-ray examination was made by Dr. D. D. Talley, and a large pituitary tumor was found which had eroded the body of the sphenoid and invaded its sinus.

Neurologic examination was made by Dr. C. C. Coleman without adding to the positive observations. He did not advise operation.

The patient was then referred to Dr. Talley for deep x-ray treatment. Dr. Talley thought that the tumor was probably malignant and through three areas gave the doses necessary for treatment of carcinoma. Under treatment the headache became distinctly less, and the patient felt improved. At the end of the first series of treatments, a slightly amber-colored, translucent, watery fluid began to drop from the patient's nose. On laboratory examination, Dr. W. A. Sheppard thought this was cerebrospinal fluid. A second series of x-ray treatment was given six weeks after the first, and the patient is now entirely free from headache.

SUMMARY AND CONCLUSIONS

A patient presented himself who had a serious, if not fatal, condition, with practically only one symptom, although an outstanding one, i. e., a definite constant headache of five months' duration which came on suddenly at the age of 57. During subsequent examinations, only two observations were made from which a diagnosis might have been deducted. The first observation was made during a complete but negative general examination and suggested tumor of the brain; the second, an objective symptom, was the condition of the sphenoid sinus as found with the probe. Although the condition was interpreted as inflammatory, correct diagnosis was obtained by roentgenography.

Up to this stage of destruction in this particular type of tumor, although the rhinologist may suspect the nature of the condition, roentgenography seems to be the only means of diagnosis. Dr. Coleman pointed to the likelihood of such a tumor eroding into the sphenoid sinus when the diaphragma sella was unusually thick, and explained this by the known fact that bone tissue frequently gives way to pressure before soft tissue. Dr. Talley explained the decrease in severity of the exacerbations of headache prior to treatment by the lowered intracranial pressure when the tumor eroded into the sinus, and it was believed that the leakage of serum was probably due to shrinking of the tumor.

Clinical Notes

A NEW INSTRUMENT FOR CATHETERIZING THE EUSTACHIAN TUBE

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The device may be clamped on the regular nasoscope or antroscope. It consists of the block which is located at the distal end and which causes the catheter to deflect either to the right or the left, according to the position of the tube which guides the catheter and which is cut slanting adjacent to the stationary block. The catheter carrier is rotatably attached to the clamping device.

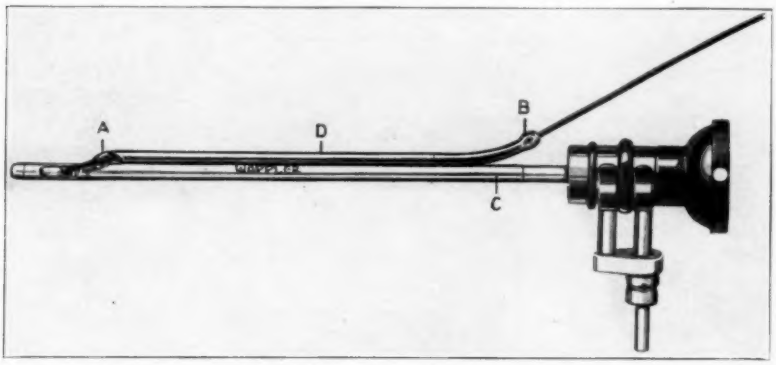

Fig. 1.—New catheterizing instrument. *A* indicates the block; *B*, the rotatable handle; *C*, the clamping device and *D*, the catheter carrier.

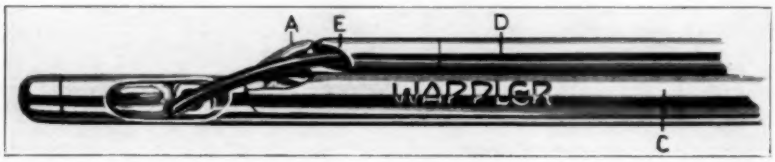

Fig. 2.—Detail of tip of catheter. *A* indicates the block; *C*, the clamping device; *D*, the catheter carrier and *E*, the obliquely cut end of the catheter carrier.

The rotatable catheter carrier has the usual bend on the near end, which in this case serves as a handle for rotating the tube to indicate the position and also as a means for regulating the degree of deflection. It is obvious that a thinner catheter could not be deflected at the same angle as a larger catheter, but by virtue of the stationary block and the bias cut end of the catheter tube and the various degrees at which the bias cut end can be placed, a greater or less deflection can be obtained. The relative position of the outer end of the tube with its curved end and the distal end can quickly be comprehended.

With reference to the direction in which the catheter is deflected, the deflection at the distal end is always toward the end opposite that of the catheter on the out-

side, so that when deflected the instrument is in the shape of the letter S. The advantage of this device is apparent, since the greatest difficulties are always encountered in getting the proper degree of deflection and of knowing certainly that the catheter will come out where it is wanted.

The mechanical advantages of this instrument are:

1. After insertion, the bougie can be deflected to the right or to the left.

2. The angular deviation of the bougie, electrode or catheter used can be definitely controlled and changed in accordance with anatomic variations in the location of the eustachian orifice.

3. It does not have a distal curve, only a straight tube and block which facilitates passage of the catheter, bougie or electrode without danger of catching in the distal curve.

4. Application is rapid, sure and under direct vision.

To sum up the clinical advantages: Advantage has not yet been taken of all the possibilities of this instrument. After ten months' experimentation in certain directions, it can be definitely stated: 1. The instrument affords easy access to the eustachian tube for topical medical applications, and for intra-eustachian manipulation. This is accomplished under direct vision and is nontraumatic. 2. It affords a means for the application of diathermy directly to the middle ear. A preliminary report on this work is soon forthcoming. 3. Ulcerations and growths in the inaccessible posterior nares can be treated under direct vision.

TECHNIC

The nose is lightly cocainized with a 10 per cent cocain epinephrine solution.

The instrument attached to the nasoscope is passed into the nose along the inferior meatus until the eustachian orifice comes into view.

If a bougie is to be inserted in the left eustachian tube, the handle of the instrument is turned to the right, and vice versa. The bougie is passed into the lumen of the instrument, and there under direct vision into the eustachian orifice. The angular deviation of the bougie may be changed to meet with the location of the eustachian orifice. The simplicity and rapidity of operation leave nothing to be desired.

Through the cooperation of Mr. Reinhold H. Wappler of the American Cystoscope Makers, Inc., this instrument has been perfected.

225 West Eighty-Sixth Street.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

ACUTE AND CHRONIC OTITIS MEDIA AND SINUS THROMBOSIS *

SAMUEL J. KOPETZKY, M.D.
NEW YORK

In reviewing the literature for 1927 on the subjects of acute and chronic otitis media purulenta and sinus thrombosis, I have selected only those papers which promote discussion. Many papers appeared which are not reviewed in these pages; but either they were of the type which reiterates well established facts or they reported cases interesting enough in themselves but not of unusual import.

ANATOMY, PATHOLOGY AND BACTERIOLOGY

In determining the effect of pneumatization of the mastoid process on the progress of an otitic infection, Wonsowski [1] arrived at the following conclusions from a study of sixty cases: He found that in a well pneumatized mastoid process a greater amount of intramastoidal destruction is presented at operation; but, on the other hand, a minimum number of complications occur when a mastoid process of this type is infected. In the diploic type of mastoid process, there is a tendency to diffusion of the infection in the interior of the bone, with the consequent formation of perisinal and epidural abscesses. A cholesteatoma is usually found in the sclerotic mastoid; but it may also be associated with any of the other types.

I believe that the observations of Wonsowski are only partially true. There is no doubt that the anatomic formation of the mastoid process has some influence on the production of an intracranial lesion; but to state that the majority of dural or vascular lesions are limited to the diploic or sclerotic mastoid is to overlook the rôle played by bacteria, by the type of pathologic lesion present, by the duration of the disease and by the pressure of pent-up pus. The fact that an infection with *Streptococcus mucosus capsulatus* is present is of infinitely more importance in the production of complications than the type of mastoid process in which the infection exists. A hemorrhagic mastoiditis is more apt to produce a blood stream infection than a coalescent mastoiditis, regardless of anatomic considerations. Similarly, an

* From the Oto-Laryngological Department of Beth Israel Hospital.

1. Wonsowski, T.: Pneumatisation de l'apophyse mastoïde et otorrhée, Rev. de laryngol. **48**:451 (July 15) 1927.

infection of the mastoid process which has been permitted to continue beyond the period of safety will cause an erosion of the inner table sooner than one which has been recognized and operated on early in its course.

I have come to look on the process of eburnation, the so-called sclerosis of the mastoidal structure, as a protective reaction to a lesion in the bone. I believe it is an effort of nature to encapsulate the lesion with compact bone. It is agreed that sclerosis will be found surrounding a cholesteatomatous infiltration of the mastoid process; but it is doubtful in the extreme that the sclerosis stands in causal relationship to the cholesteatoma.

Studying the pneumatization of the mastoid process from a roentgenologic point of view, Brock [2] stated that a chronic purulent otitis media, due either to cholesteatoma or to a perforation of the attic, is never seen in a normally pneumatized mastoid process. He felt that a typically normal membrana tympani is conclusive proof of a normally pneumatized mastoid. He expressed the belief that any condition of the tube or middle ear which prevents aeration of the tympanic cavity may be the cause of nonpneumatization. In sixty cases of unilateral chronic purulent otitis media, both mastoids were identical in structure on the x-ray plate. Brock credited Witmaack, Moirret and Heine with the statement that filling in of the mastoid cells with bone following inflammation is impossible and that eburnation of the mastoid process is not the result but the cause of suppuration.

A great many of Brock's statements are not in accord with my experience and that of many others. In the first place, I have seen any number of perforations of the attic in the exanthems in which the mastoid process was thoroughly pneumatized, both on the x-ray plate and at operation. If one realizes that a perforation of the attic in an acute case is an early sign of chronicity, it becomes conceivable that the same perforation will be found at a later date with a mastoid process which has become sclerotic. Furthermore, I do not comprehend how an eburnized mastoid can cause suppuration or even contribute to its existence. Whenever suppuration of bone exists, osteoclasis is followed by osteogenesis. It is true that such conditions only occur in a chronic infection of the bone; but that is because in an acute infection the systemic absorption and other symptoms are so apparent that surgical intervention is recognized as imperative. Only in cases in which repeated attacks of acute mastoiditis have occurred, in each of which healing has taken place without surgical intervention, is the gradual replacement of the cellular structure by compact bone perceptible. In such cases roentgenograms taken at each attack will show increasing density and less cellular structure each time; and finally, when operation

2. Brock: Trommelfellbild und Pneumatisation des Warzenteils; eine rontgenologische Studie, Tr. Deutsche Otol. Gesellsch., Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:241 (Nov.) 1926.

is undertaken, more or less dense bone is found to have replaced the mastoid cells.

Keen [3] made an interesting observation concerning the structure and the pathologic changes of the ossicles in persons with chronic purulent otitis media. He found that the point of attack on the malleus is at that portion of the head which is away from the incus. From this point, caries proceeds to the hammer handle. In caries of the incus, the tip of the long process is affected first. The disease then progresses upward to the body of the incus. The short process is generally well preserved. Incidentally, Keen stated that when the long process of the incus is found destroyed at operation, it is safe to curet the inner wall of the middle ear, since the stapedial crura will also have been destroyed by necrosis. He believed, from his observations and those of Kikuchi,[4] that it may be accepted as fact that the middle ear presents full adult dimensions at birth and that further growth does not take place. What occurs are merely adjustments in the axis of the drum due to the growth of the external auditory canal and the mastoid process. On examination and comparison of the ossicles of adults and infants, difference in size or contour cannot be noted.

Further studies of the significance of the von Schilling white cell count were made by Hesse.[5] He is in accord with the observations of Weiss, Levy and Heidemann, which were reviewed last year, that this method of counting the cells gives more information than the total leukocyte count alone. His observations also bear out my observation that the count is more valuable as a prognostic than as a diagnostic aid. He found that a well defined shifting to the left is more marked in persons with sinus thrombosis than in those with meningitis and abscess of the brain.

Wirth [6] reported a case of acute purulent otitis media followed by acute mastoiditis due to a mixed infection of anhemolytic streptococcus and *Bacillus pyocyaneus*. Each of these organisms has but slight pathogenicity for man and practically none for mice. Yet, in a mouse, the mixed infection led to a fatal sepsis. It seems that in conjunction with other microbes causing otitis, *Bacillus pyocyaneus* lends invasive qualities to the other organism. I have noted this in my own practice and have found that patients who present the pyocyaneus bacillus as a secondary or primary invader frequently develop erysipelas as a

3. Keen, J. A.: Ossicles in Middle Ear Suppuration and Their Comparative Size in Children and Adults, J. Laryng. & Otol. **42**:174 (March) 1927.

4. Kikuchi, J.: Beitrage zur Anatomie des menschlichen Amboss, Ztschr. f. Ohrenh. **42**:122, 1903.

5. Hesse: Das Blutbild und die Entstehungsbedingungen von Komplikationem im Anschluss an Mittelohreiterungen, zugleich ein Beitrag zur Klinischen Verwertung des Blutbildes, Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:232 (Oct. 6) 1926.

6. Wirth, E.: Subakute Mastoiditis durch Mischinfektion von B. Pyocyaneous und Streptococcus anhaemolyticus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:188 (Jan. 31) 1927.

secondary complication following operations on the mastoid. Five such cases have been reported by Almour and myself.[7]

An interesting method for the differentiation of otitic suppuration due to necrosis of the bone from that caused by infection of the soft parts has been presented by Friesner and Rosen.[8] The test is based on a microcolorimetric estimation of the calcium content of the pus from the middle ear. After examination of pus from all sources by this means, the authors have arrived at a maximum calcium content for pus resulting from suppuration of soft parts. When chemical examination shows the calcium content to exceed this maximum, it is presumed that bone also is involved. The authors feel that this test may become a valuable aid in the diagnosis of destruction of bone due to suppuration in the mastoid process. In this way, it would help more accurately to determine the time for operative intervention, especially in cases which are on the clinical borderline. More experience with this procedure will be necessary before it is accepted as a criterion for the diagnosis of destruction of bone and a definite indication for surgical intervention.

ACUTE AND CHRONIC OTITIS MEDIA

The investigation of the systemic effects of infection of the mastoid in infants still holds considerable interest for the otologist and the pediatrician. Alden,[9] Dean,[10] Lierle[11] and Lyman[12] determined that there is a distinct clinical entity in infants in whom the only otologic signs are a downward sagging of the superior canal wall just external to the annulus but in whom constitutional evidences of absorption are the predominating clinical features. The latter consist of rapid loss in weight, fever, diarrhea, dehydration, vomiting and acidosis, due to the rapid and massive absorption of toxic products. In this group of cases, a subperiosteal abscess is rarely found; and one should not wait for its appearance before performing an antrotomy. The clinical

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7. Kopetzky, S. J., and Almour, R.: Erysipelas following Bacillus Pyocyaneous Infection in Mastoid Wounds, Am. J. Surg. **2**:589 (June) 1927.

8. Friesner, I., and Rosen, S.: Calcium Content of the Pus; Comparison of Discharge from Ears with and without Bone Destruction, J. A. M. A. **88**:1231 (April 16) 1927. Friesner, I., and Rosen S.: A New Aid in the Diagnosis of Mastoiditis, Laryngoscope **37**:707 (Oct.) 1927.

9. Alden, A. M.: Mastoid Infections in Infants, Arch. Otolaryng. **5**:39 (Jan.) 1927.

10. Dean, L. W.: Acute Otitis in Infants, Arch. Otolaryng. **6**:201 (Sept.) 1927.

11. Leirle, D. M.: Otitis Media in Infants, Ann. Otol., Rhin. & Laryng. **36**:604 (Sept.) 1927.

12. Lyman, H. W.: Infantile Mastoiditis with Gastro-Intestinal Symptoms, Arch. Otolaryng. **6**:526 (Dec.) 1927.

symptoms in these patients have been borne out by gross observations at autopsy. Dean found that in these cases of infantile mastoiditis there is frequently a coexisting nasal sinusitis. He felt that it is as important to treat the latter as it is to care for the mastoidal infection, since it is difficult to decide which condition is more influential in the production of the systemic disturbance.

Babcock [13] felt that when one finds a gastro-intestinal disturbance in a child, associated with a membrana tympani which is dull or creamy white, it is advisable to maintain aural drainage by repeated paracentesis and only as a last measure to resort to mastoidectomy. He considered vaccines and actinotherapy valuable in the treatment of such patients. I cannot agree with Babcock as to the preferability of repeated paracentesis to simple mastoidectomy. Repeated trauma to the drum is certain to result in some loss of acuity of hearing. In the early days of life, when it is essential to maintain all the senses at their highest functional level, simple mastoid operation which affords certain drainage and preserves the hearing at the same time is far more advisable than repeated paracentesis. I do feel, however, that an incision in the drum, placed so as to include part of the superior canal wall, should be made before mastoidectomy is undertaken in these cases.

The reports of the condition found at operation in these cases leave much to be desired. The postembryonal tissue which is present in the middle ear and adnexa of the infant is too easily mistaken at operation for edematous, swollen, infiltrated and infected tissue. In other words, the cases in which operation was performed which make up the published reports lack the pathologic proofs, adduced after competent microscopic observation, which are necessary to establish firmly the stand taken by the proponents of antrotomy in the infant in this type of disease.

The report of Lefler [14] is of considerable importance as an indication of what can be accomplished in the prevention of chronic purulent otitis media due to scarlet fever. She advocated early paracentesis in all infections of the ear occurring during scarlet fever, with strict asepsis before and after incision. When, after incision or spontaneous rupture, the discharge is protracted, a modified radical operation is undertaken. In this way, hearing is preserved. Over the period of one and one-half years which this report covers, every patient treated in this manner has been discharged with a dry ear and an intact drum.

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13. Babcock, E. S.: Chronic Otitis Media in Infancy, Arch. Pediat. **44**:508 (Aug.) 1927.

14. Lefler, A. B.: Scarlet Fever Otitis, Ann. Otol., Rhin. & Laryng. **36**:495 (June) 1927.

Lefler compared these results with those of Ross,[15] who concluded that in 15 per cent of all patients with chronic purulent otitis media the condition is due to scarlet fever and in 10 per cent of all deaf mutes it can be attributed to the same disease.

Costen[16] reported two cases of diphtheritic otitis media which were virulent. The ordinary culture did not reveal the type of infection. It was only when cultures were made from the aural discharge on the special (Loeffler's) medium that the causative organism was isolated.

Schlittler[17] summarized the cases of mastoiditis which occurred during the epidemic of grip in 1927. These cases were characterized by severe pain and septic temperature. Twenty-two per cent of the cases (193) were of the hemorrhagic type; in 33 per cent complications (sinus thrombosis, meningitis and labyrinthitis) developed. The predominating organism was *Streptococcus hemolyticus*. Next in frequency of occurrence was *Streptococcus mucosus* and the pneumococcus. This is in accord with the experience of the United States during the influenza epidemics of 1918 and 1920.

TUBERCULOSIS OF THE MIDDLE EAR

Blegvad[18] described thirty-five patients with tuberculosis of the middle ear who were seen and watched by him from the onset of the disease. Pain was not present at first, but the deafness was noticeable. Slight swelling and dulness of the drum were followed by some redness. The vessels were not injected at the periphery of the drum. The radial vessels, however, were engorged. The membrane gradually swelled, and the short process was obliterated. The drum took on a yellowish color. Perforation then occurred, and a chronic purulent otitis media resulted.

Lussmann and Bendove[19] found that of forty-one cases of chronic purulent otitis media in tuberculous patients only three showed acid-fast bacilli in the discharge; and the pus from only four others produced tuberculosis in the guinea-pig. Yet only six patients failed to improve under treatment particularly directed toward clearing up the tuberculosis. All others showed a marked anatomic improvement; and in seven instances functional restoration of the ear was noted.

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15. Ross, E. L.: Otitis Media in Scarlet Fever, Ann. Otol., Rhin. & Laryng. **33**:1319 (Dec.) 1924.

16. Costen, J. B.: Diphtheria Infection of the Middle Ear and Mastoid: Report of two cases. Arch. Otolaryng. **5**:119 (Feb.) 1927.

17. Schlittler, E.: Ueber die akute Mittelohrentzündung der Grippewelle des Frühjarhres 1927, Schweiz. med. Wchnschr. **8**:1042 (Oct. 29) 1927.

18. Blegvad, N. R.: La tuberculose de l'oreille moyenne chez les phtisiques, Acta Otolaryng. **10**:513, 1927.

19. Lussmann, F. J., and Bendove, R. A.: Chronic Otitis Media in the Tuberculous: Local Ultraviolet Light Treatment, Arch. Otolaryng. **6**:153 (Aug.) 1927.

Spencer [20] emphasized the importance of heliotherapy in accomplishing an ultimate cure of a tuberculous patient with suppurative disease of the middle ear.

In this connection it is interesting to note that Stern,[21] in a statistical study of 5,380 patients suffering from chronic disease of the middle ear, found only 0.65 per cent to be due to tuberculosis. The most important etiologic factor in the production of chronic suppuration of the middle ear was a diseased condition of the nasopharynx and sinuses, which accounted for 59.9 per cent of the cases. The infectious diseases of childhood were responsible for only 3.7 per cent of the cases studied. This observation is not in accord with that of Ross previously mentioned.

Both Bernfeld [22] and Beck and Pollack [23] stressed the influence of diabetes on the course of a mastoidal infection. Early recognition of the mastoidal infection, operation under local anesthesia and vigorous antidiabetic therapy will tend to ward off the complications which are so prevalent in this type of mastoiditis.

TREATMENT

Wirth [24] studied the effect of boric acid on fifteen varieties of organisms in vitro. The growth of the organisms was impeded considerably when a 2 per cent or 4 per cent solution of boric acid was added to Schottmüller's blood agar or blood bouillon. The germicidal effect of boric acid was less noticeable. It likewise had little effect on the virulence of the organisms. Wirth felt, however, that in vivo the pharmacologic qualities of the boric acid have a more active disinfectant action.

Guttman [25] studied the effect of Calot's solution on eighty-one persons with chronic otorrhea. Thirty-one patients with the nondangerous type of the disease were all cured. Thirty-five of the thirty-eight with the dangerous type of chronic purulent otitis media were cured. None

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20. Spencer, F. R.: Tuberculosis of the Middle Ear, Arch. Otolaryng. **6**:242 (Sept.) 1927.

21. Stern, M.: Die Otitis media chronica in den Jahren 1911-1921 an der Universitäts-Ohrenklinik in Frankfurt a. M., Ztschr. f. Hals-, Nasen- u. Ohrenh. **16**:162 (Sept. 22) 1926.

22. Bernfeld, K.: Mastoiditis bei Diabetes mellitus, Monatschr. f. Ohrenheilk. **60**:980 (Oct.) 1926.

23. Beck, J. C., and Pollack, H. L.: Pathologic Conditions of the Ear, Nose and Throat in Diabetes, Arch. Otolaryng. **5**:400 (May) 1927.

24. Wirth, E.: Untersuchungen über die wachstumshemmende, keimtötende und virulenzherabsetzende Wirkung der Borsäuer auf die Erreger der akuten und chronischen Otitis media, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:499 (March 18) 1927.

25. Guttman, M. R.: Chronic Otorrhoea: Its Response to Calot's Solution, Illinois M. J. **52**:143 (Aug.) 1927.

of the twelve patients with cholesteatoma were affected in any way. Calot's solution is a combination of guaiacol, 1 Gm., creosote, 5 Gm., ether, 30 Gm., iodoform, 10 Gm. and olive oil, 70 Gm. Treatment is by the drop method.

Wonsowski,[26] on the other hand, did not obtain results from the use of Calot's solution other than a lessening of the foul odor. In thirty-two patients with chronic purulent otitis media whom he treated with it healing was not observed.

In a series of 110 cases of chronic purulent otitis media, Horn [27] obtained a dry ear and an improvement in hearing in 50 per cent of the cases by correction of the nasal pathologic condition, attention to the general health and packing the canal down to the middle ear with a 20 per cent solution of collargol ointment. The last named, which is a silver preparation, is employed every second day.

Hazeltine [28] recommended inflation as an adjuvant in the treatment for acute purulent otitis media. He felt that only in this way can the pus from the region of the attic and the eustachian tube be helped to find an exit through the perforation in the drum. He considered that this method of treatment, together with the routine therapy to the ear, will do more to avoid the occurrence of a mastoid suppuration than any other means of treatment.

Reese [29] advocated ossiculectomy in patients with chronic suppurative otitis media in whom the condition does not yield to treatment and in whom urgent need for the radical operation does not exist. Of six patients so treated, three were cured and in three the suppuration continued. Reese suggested this as a preliminary procedure before the radical mastoid operation is undertaken.

Gilhespy [30] reported his results from the operative treatment for chronic purulent otitis media in children. A simple mastoid operation was performed in nine cases, in six of which dry ears resulted. One failure was due to the presence of a cholesteatoma. A conservative radical operation was performed in ten cases and a dry ear obtained in all. The indication for the latter procedure was the presence of a perforation of the attic. In such cases, Gilhespy considered a conservative radical operation the best. The outer wall of the attic is

26. Wonsowski, T.: Ueber die Calotsche Lösung bei der eitrigen Mittelohrentzundung, Monatschr. f. Ohrenh. **61**:921 (Aug.) 1927.

27. Horn, J.: Conservative Treatment of Chronic Otitis Media Purulenta; Preliminary report, M. J. &·Rec. **125**:721 (June 1) 1927.

28. Hazeltine, B.: Tubotympanic Treatment in Acute Otitis Media, J. Ophth. Otol. & Laryngol. **31**:40 (Feb.) 1927.

29. Reese, M. D.: Ossiculectomy in Treatment of Chronic Purulent Otitis Media, Laryngoscope **37**:121 (Feb.) 1927.

30. Gilhespy, F. B.: Report on the Operative Treatment in Children with Chronic Suppurative Otitis Media, J. Laryng. & Otol. **42**:520 (Aug.) 1927.

removed and the mastoid cavity lined with skin from a flap in the posterior membranous canal wall. When the radical mastoid operation was performed, Gilhespy obtained a dry ear in nine of the seventeen patients who were operated on.

SINUS THROMBOSIS

Deutsch [31] noted that metastatic lesions are more apt to occur in persons in whom sinus thrombosis follows an acute otitis than in those with periphlebitis subsequent to chronic aural disease. The greater percentage of patients in whom sinus thromboses followed acute purulent otitis died; whereas the greater percentage of those in whom sinus thromboses followed a chronic purulent otitis recovered. Deutsch also stated that when the wall of the sinus was normal in appearance in the patients with sinus thrombosis due to acute suppuration, the prognosis was poor, as most of those patients died. When granulations were noted over the wall of the sinus, the patient was more apt to recover. Deutsch's observation that sinus thrombosis following acute mastoiditis must have a different significance from sinus thrombosis following chronic mastoiditis is not new. All who are familiar with the pathogenesis of sinus thrombosis will realize that in persons with chronic mastoiditis the wall of the sinus is gradually compressed once the inner table is eroded. In this way, the blood stream is slowed and the channel is narrowed. When the intima is injured a sterile thrombus forms, and this is soon organized so as to obliterate the vein. This accounts for the frequent presence of an aseptic thrombus within the sinus at the radical operation for chronic purulent otitis. If this thrombus should become secondarily infected from an acute infection superimposed on the old chronic infection, fluid pus will be found in the sinus. The sinus, however, has been sealed off by the obliterating thrombus and rarely permits any bacteria to enter the general circulation. In addition, the preexisting tolerance to the chronic infection and the establishment of a protective thrombus before the advent of the acute infection afford a prepared resistance to the new infection and consequently a better prognosis.

It is unusual to find a normal wall of the sinus in persons in whom acute coalescent mastoiditis is complicated by sinus thrombosis. The presence of granulations most frequently marks the site of the periphlebitis. These granulations are protective in nature, as is the thrombus which subsequently forms within the sinus. Since the body has already furnished protection against the invasion of the sinus by bacteria, a better prognosis is to be expected than in the patients with hemorrhagic mastoiditis in whom the wall of the sinus is normal in appearance

31. Deutsch, L.: Beitrag zur Klinik und Pathologie der Sinus Thrombose, Wein. klin. Wchnschr. **40**:713 (June 2) 1927.

because the infected thrombus entered it by direct extension from a smaller vessel in the bony intercellular walls.

As a preventive measure to avert the onset of septic sinus thrombosis in persons with acute coalescent mastoiditis, Almour [32] recommended obliteration of the sinus when an extensive periphlebitis is found, regardless of whether or not systemic signs are present. He felt that since ill effects do not follow surgical obliteration of the sigmoid sinus, it is far better to eradicate a focus of infection which is potentially capable of producing septic sinus thrombosis. In his paper, he also emphasized the importance of unilateral headache, a persistent polymorphonucleosis and an appearance of well being as characteristic early signs of sinus thrombosis; and he relied more on these symptoms than on the results of blood culture.

In a series of well balanced charts, Swift [33] differentiated sinus thrombosis from meningitis and abscess of the brain. His acceptance of a septic temperature as a constant sign of sinus thrombosis, however, is not borne out by the clinical experience of others.

Ottenberg [34] presented a new procedure for the diagnosis of sinus thrombosis. It consists of taking simultaneous blood cultures from both internal jugular veins. He did not reach any conclusions as to the interpretation of the results of the procedure; for, although in some cases the greater number of colonies was found on the side of the thrombosis, in other instances the greatest growth was obtained from the internal jugular vein opposite the site of the lesion. In cases in which the greatest growth is obtained from the opposite side, he felt that the lower end of the affected sinus must be blocked and that the organisms have found their way through the torcula because of a reversal of the current after the obliterating thrombosis had occurred. For this view to hold, it must be assumed that both lateral sinuses arise from the torcula, but it is definitely established that the superior longitudinal sinus often empties into one lateral sinus, whereas the inferior, straight and occipital sinuses empty into the other lateral sinus and a connection does not exist between the lateral sinuses on the right and left sides. The procedure is interesting; but it by no means affords as accurate a method of differentiation as a positive Tobey-Ayer test. Neither the rationale nor Ottenberg's deductions from his observations are conclusive.

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32. Almour, R.: Prevention and Minor Symptoms of Sinus Thrombosis, Arch. Otolaryng. **6**:373 (Oct.) 1927.

33. Swift, G. W.: The Differential Diagnosis between Septic Meningitis, Brain Abscess and Lateral Sinus Thrombosis Complicating Mastoiditis, Ann. Otol. Rhin. & Laryng. **36**:669 (Sept.) 1927.

34. Ottenberg, R.: The Diagnosis of Lateral Sinus Thrombosis: New Procedure, Arch. Otolaryng. **5**:43 (Jan.) 1927.

In this connection, Druss [35] reported a case of primary thrombosis of the jugular bulb in which the diagnosis was made by utilizing the differential blood culture from both internal jugular veins. The culture from the opposite vein yielded 275 colonies to the plate, whereas the culture from the affected side showed only nine colonies. An obliterating thrombus was found. In such cases, it would be of great interest if the reports of successive blood cultures were published, in order to correlate the condition found on culture and the clinical picture at various times during the course of the case.

McDaniel [36] reported a case of primary thrombosis of the bulb which was typical in every respect. The mastoid process was normal at operation, the blood culture was positive and the patient had numerous metastatic abscesses.

Wieder and Bates [37] reported a case of primary thrombosis of the bulb in which the mastoid process was found normal. They found that the daily use of Pregl's iodine solution intravenously is of marked benefit in streptococcemias. Twenty cubic centimeters of the solution is injected every day.

Roberts [38] cited a case of otorrhea, followed by sepsis and a positive blood culture of *Streptococcus hemolyticus,* in which the roentgenograms showed a normal mastoid process. The child developed an infection of the foot, and thereafter the blood culture became sterile. The patient was not operated on and recovered. Such cases are interesting only as exceptions. It is well known that a metastatic lesion in the soft parts will usually clear the blood stream of all bacteria. But to risk leaving the original focus of infection in situ is too grave an undertaking.

Schlander [39] deprecated the utilization of the bulb operation before conservative measures are employed. In cases of sinus thrombosis in which a continued suppuration from the bulb is evident, one should wait before resorting to a bulb operation. When thrombosis of the posterior condylar vein is present, the operation is certainly not of value in stopping the source of continued sepsis. It is only in those cases in which osteitis of the jugular fossa is present that the bulb operation will be successful in eradicating the diseased bone. When

35. Druss, J.: Primary Jugular Bulb Thrombosis Presenting Certain Unusual Features, Laryngoscope **37**:439 (June) 1927.

36. McDaniel, F. C.: Jugular Bulb Thrombosis; Multiple Abscesses; Recovery, Laryngoscope **37**:428 (June) 1927.

37. Wieder, H. S., and Bates, W.: Primary Jugular Bulb Thrombosis with Numerous Metastatic Infections; Operation; Recovery, Laryngoscope **37**:48 (Jan.) 1927.

38. Roberts, E. R.: Septicemia of Otitis via Jugular Bulb, Arch. Otolaryng. **6**:383 (Oct.) 1927.

39. Schlander, E.: Zur Therapie der otogenen Bulbusthrombose, Wien. med. Wchnschr. **77**:1105 (Aug. 20) 1927.

abscesses of the neck appear due to the bulbar suppuration, Schlander found it sufficient to incise and drain the abscess in order to effect a cure.

Hofer [40] discussed the occurrence of sinus thrombosis in infants. He stated that the clinical picture presented by this disease in infants is more like that found in meningitis. A high continuous temperature, rigid neck and other signs of meningeal irritation are present. One is apt to overlook the real cause of the sepsis because of the rapid metastatic lesions, particularly in the lungs and the long bones. The secondary pneumonia or osteomyelitis is mistaken for the primary disease.

Lillie [41] reported three cases of thrombosis of the superior longitudinal or straight sinus by extension from the lateral sinus. The cases all started with chronic purulent otitis media. The outstanding symptoms were repeated chills, severe headache and continued temperature. The first and third cases were verified at autopsy and the second case at operation. Lillie felt that operation on the superior longitudinal sinus should be undertaken in these cases. This observation is extremely important and might possibly explain the continuation of the clinical picture of sinus thrombosis after a thorough exenteration of the thrombus in the sigmoid sinus and the obtainment of a sterile blood culture.

The observation of Fremel [42] is also important. He reported two cases of chronic purulent otitis media due to cholesteatoma in which sepsis subsequently developed. Thorough inspection of the lateral sinus did not reveal a thrombus but at the entrance of the superior petrosal, at the knee, a formed thrombus was seen to protrude from the petrosal. The petrosal was exposed by removing the inner table, and the thrombus was evacuated. Bleeding was controlled by gauze plugs.

51 West Seventy-third Street.

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40. Hofer, I.: Zur Symptomatologie der otogenen Pyämie im Säuglingsalter, Wien. med. Wchnschr. **77**:221 (Feb. 12) 1927.

41. Lillie, H. I.: Thrombosis of the Superior Longitudinal or Straight Sinus by Extension from the Lateral Sinus: Report of Three Cases, Ann. Otol., Rhin. & Laryng. **36**:324 (June) 1927.

42. Fremel, F.: Ueber isolierte Erkrankung des Sinus petrosus superior, Monatschr. f. Ohrenh. **61**:409 (June) 1927.

Abstracts from Current Literature

Ear

From the Proceedings of the German Otolaryngologic Congress, 1927

NOISY OCCUPATION AND THE EAR. BECK, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:387, 1927.

The author undertook a systematic investigation of workers engaged in noisy occupations. He established as a standard measure of noise the intensity that could just be heard over the telephone. This he calls one "wien," which is the threshold of hearing. For convenience of classification, he then has another measure called a "phon." Two wiens equals 1 phon, 4 wiens equals 2 phons, 8 wiens equals 3 phons, and so on by logarithmic progression. His experiments show that occupations in which there was noise of about 9 phons caused deafness, and that the deafness advances in time and severity in direct proportion to the intensity of the noise. Less than 9 phons of constant noise do not appear to cause deafness.

The following occupations listed by him are some of those that are detrimental to hearing: sharpening wheel for iron tools, 10 phons; textile factories (weaving), from 10 to 11 phons; revolving drum in cement work, 11 phons; coal mill in cement work, 11 phons; one wire factory, from 12 to 13 phons; boiler factory, 14 phons.

An interesting observation is that the noise of a running locomotive is from 6 to 9 phons—below the harmful noise. Locomotive engineering has been considered a prolific cause of deafness.

INJURIES OF THE EAR IN MINERS. MAUTHNER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:392, 1927.

The author calls attention to the frequency of injuries to the head in miners, and especially to the occurrence of such injuries caused by crushing or compression, because of the narrow spaces in which they work. He thinks that these injuries should be considered a clinical entity. According to the severity of the trauma, the injuries may range anywhere from those of the external ear to fracture of the skull. In his experience, the characteristic lesion is fracture of the upper posterior part of the canal wall, including the margin of the membrani tympani, in contradistinction to the fracture of the anterior wall caused by blunt force at the jaw. Paralysis of the facial nerve may be due to hemorrhage in the canal or to fracture. Clinical cases are reported.

OCCUPATIONAL DEAFNESS. PEYSER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:397, 1927.

The author makes a plea for the prevention of occupational deafness, and describes a number of inventions that have aided in this prevention, for instance, welding as a substitute for riveting. He announces the foundation of a society for this purpose.

EXAMINATIONS IN HUMANS FOR OTOLITH FUNCTIONS. GRAHE, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:411, 1927.

The vestibule consists of two anatomic and physiologic parts: (1) the saccule and utricle with the otoliths, which sense the head position at rest; (2) the canal system which senses the movement of the head in space. The author has devised a table to test the function of the otolith. The patient is strapped to the table, which can be rotated in either the coronal or sagittal plane. The sense of vertical

position is tested by measuring the degree of deviation from vertical that the patient can detect. The variation of normal persons is from one to three degrees in the coronal plane, and is the same to both sides. In pathologic conditions, it is considerably less on the affected side. In this plane, it is the saccule that is affected. In the saggittal plane, impulses from both utricles cooperate. The normal error in this plane may be seventeen degrees.

The head erect reflex was demonstrated in animals by Magnus and DeKleyn. In this test, the body is strapped and the head is free. The table is tilted in the coronal plane, and the head automatically assumes the upward position. The test is not so reliable, and as the patient unconsciously may interfere, it is necessary to have him blindfolded and his attention distracted by some arm movement or other means. The test is more reliable in infants. The results of this test were questionable in eight of thirty-seven cases; in eleven, the results were normal. In fifteen of eighteen pathologic cases, the reflex was weak or absent; in three cases it was perverted.

A spontaneous deviation of the head is frequently seen. It is an inclination toward the affected side, sometimes associated with slight turning. There are many causes for it, but it is more marked on the table.

For testing the rotation of the eyes, a spectacle frame is used with a hair line crossed center on the center of the pupil; over the other eye there is an opaque glass with a transparent cross on the center of the pupil. A spot coinciding with one of the lines is marked on the cocainized cornea with a barium gelatin pencil. The degrees are marked on the rims or the periphery of the lenses. In this way, it is possible to detect minute degrees of rotation.

THE FUNCTIONAL SIGNIFICANCE OF THE ROUND WINDOW. KRAINZ, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:433, 1927.

The round window is compensatory for movement of the labyrinth fluid. It is more movable than the oval window. The hearing is affected in proportion to the limitation of movement. Interference with this movement gives the hearing reactions of nerve deafness, but hearing is improved by inflation, if the condition is due to a removable exudate at the round window. It can also be tested by the Gelle test. Deafness caused by compression of the air in the middle ear is relieved by anything that restores the air balance if the round window is functioning. The Gelle test is indicative of this. If, then, improvement occurs on inflation (in the presence of normal hearing for low tones showing normal movement of the ossicles), it is evidence that round window disturbance was present, which was removed by inflation, and treatment by inflation is indicated.

CLINICAL EXPERIMENTAL INVESTIGATION OF THE RELATION BETWEEN ENDOLYMPH PRESSURE AND HEARING. KEREKES, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:458, 1927.

Various neurologic conditions and cases of hypertension and hypotension were studied. The following conclusions were arrived at, the voice, whisper and bone conduction being the principal criteria:

1. Reduction of high blood pressure can cause improvement in hypesthesia acoustica.
2. Increased intracranial pressure is attended by shortening of bone conduction.
3. The alteration of hearing goes hand in hand with lowering of intracranial pressure.
4. The foregoing points indicate that normal hearing requires optimal intralabyrinthine pressure.

PICTURES OF THE MEMBRANI TYMPANI ENLARGED FROM TEN TO THIRTY TIMES. LUESCHER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:485, 1927.

The author describes an ear microscope with head rest and electrical illumination which gives much greater detail than is possible with the loupe. Many details are

described; for instance, it is possible to see the blood flow, which varies from time to time in different people. Various other conditions are described: acute otitis media, herpes and traumatic changes. The author believes this improvement in examination may have forensic importance when further experience establishes the significance of the changes seen.

NEW IDEAS REGARDING THE PHYSIOLOGIC EFFECTS OF POOR VENTILATION OF THE EUSTACHIAN TUBE, AND SUITABLE TREATMENT. GYERGAY, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:529, 1927.

The author examined specimens with so-called tubal obstruction and did not find any permanent changes. He believes that deafness is due to the effect of poor ventilation on the muscles and nerves, and recommends massage, electrical simulation and special exercises, such as yawning and swallowing, in addition to the formerly accepted treatment. He claims to have had favorable results after the usual methods had failed. His technic is described in detail.

LEWY, Chicago.

Pharynx

From the Proceedings of the German Otolaryngologic Congress, 1927

EXPLANTATION OF TONSIL TISSUE. KELMEN, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:534, 1927.

Tonsils were removed from adolescents by the Sluder method and parts selected away from the surface and the crypts. They were kept alive as long as seven days in human plasma.

The tissue changes rapidly to the embryonal form. After the first few days, the colonies of bacteria at the margin disappear, indicating a bactericidal influence of the tonsil tissue.

The reaction of the tonsil tissue to plasma is different from other tissues; particularly, it is different from lymphatic tissue.

OPERATIVE CURE FOR TONSILLAR SEPSIS. KNICK, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:546, 1927.

The author refers to the high mortality in cases of tonsillar sepsis. Of fifty reported cases, cure was obtained in ten. The author reports in detail a case of his own in which a thrombus was found in the vena facialis communis, extending into the vessels anterior and posterior and the venae tonsillares and ascending pharyngeal and even to the vein from the parotid gland, and the communicating veins to the front of the neck. The jugular vein was tied, and the vena facialis communis was incised.

He believes the jugular vein should not be tied, unless thrombosed, but the dissection should uncover the paratonsillar region, and all thrombosed veins should be incised.

The symptoms are: tonsillitis, followed after a week or ten days by chills, intermittent fever and the blood and general clinical picture of sepsis; also signs of metastasis, such as cough and muscle and joint pains. It is not believed that tonsillectomy is desirable at this time, as it interferes with nourishment, and the sepsis is beyond the tonsils.

Paravertebral anesthesia is used.

PYEMIA FOLLOWING ANGINA. CLAUS, Ztschr. f. Hals-, Nasen-u. Orenh. **18**:557, 1927.

It may be necessary to uncover both jugular veins if the external signs do not disclose which side is affected. Claus emphasizes the necessity of following the veins into the tonsillar region, as there are frequently small pus pockets there.

He says that the tonsils should be removed as soon as the operation on the neck is finished. He reports four cases, with one death. In one case, there was retrograde thrombosis of the jugular vein to the torcular.

Uffenorde believes that sepsis as a rule is due to lymphogenous extension, thence to the larger veins.

Zange disputes this, and says that the evidence is in favor of venous origin in most of the cases. He also takes the stand that immediate tonsillectomy should be performed, as there may be small pus pockets behind the tonsil, as reported by Frankel, which may cause further trouble if not drained.

Hofer reports on a large amount of clinical material in the Hajek Clinic, and believes that infection takes place directly through the tonsillar veins and indirectly through the lymph glands.

LEWY, Chicago.

Larynx

From the Proceedings of the German Otolaryngologic Congress, 1927

CHARACTERISTIC OF MUTATION. SILBIGER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:500, 1927.

The author quotes the Biedl classification: (1) pubescence or prepuberty, (2) adolescence, (3) maturity.

During the period of rapid growth, there is endocrine disharmony with hyperactivity of the thyroid gland and anterior lobe of the hypophysis. The gonads have not yet assumed their important lead in the endocrine harmony. During the period of rapid growth of cartilage and bone, the laryngeal cartilages grow, the sagittal diameter of the larynx increases, and there is muscular hypotonus. On attempted phonation, the chords flutter instead of vibrate, and there is defective closure posteriorly. Something like that in the whispering position. The boy's voice is either hoarse or falsetto. Aside from vocal training, other devices preventing contraction of the cricothyroid muscle are useful. By hooking the fingers under the thyroid cartilage and pulling forward and upward, contraction of the cricothyroid is counteracted, and the falsetto voice is prevented. Sometimes one attempt is sufficient to cure the falsetto voice.

The administration of iodine is advised to hasten involution of the thyroid. Cases due to nondevelopment of the gonads are not hopeful.

LARYNGEAL TUBERCULOSIS IN ABACILLARY PHTHISIS. BLEGVAD, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:563, 1927.

In the government tuberculosis hospital in Copenhagen in eleven years, there were 17 patients in whom tubercle bacilli were never found among 921 with clinically certain laryngeal tuberculosis. The author recognizes that the finding of bacilli depends on the number and thoroughness of the examinations; nevertheless, there are two forms of tuberculosis: those in which bacilli are frequently found and those in which they are seldom or never found.

As to whether bacilli, when found, originate in the lungs or in the larynx or both, is not determined. Of his seventeen abacillary patients, there were several who had pronounced ulceration. Biopsy was performed in four cases and still bacteria were not found. Incidentally, fifteen patients who had bacilli became free from them with the onset of ulceration in the larynx. The percentage of cures among the abacillary cases is much higher than the average.

While hematogenous infection of the larynx is not excluded, the author believes that the large majority of cases are sputogenous. He shows that in human beings the favorite site of infection is the posterior commissure, which receives the sputum while the patient is in the lying position, while in cows it is the anterior commissure, again in the lowermost part. In horses, which seldom cough, laryngeal tuberculosis is rare.

LEWY, Chicago.

Nose

From the Proceedings of the German Otolaryngologic Congress, 1927

CLINICALLY IMPORTANT OBSERVATIONS OF THE VARIABLE ANATOMY OF THE ETHMOID. HALLE, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:504, 1927.

A number of specimens were shown illustrating the relation of the cribriform plate to the roof of the ethmoid labyrinth. In some, it is flush; in others it is below, and in some above, the roof; in others, it is different in the two sides. The author calls attention to the great danger of injuries to the dura in the region of the cribriform plate as against the relatively small danger to the dura on the outer side of the middle turbinate, and therefore advises always preserving the middle turbinate as a guide.

In the discussion, Hajek says one must proceed carefully, at all times being guided by frequent use of the probe.

INTRANASAL FRONTAL SINUS OPERATION OF HALLE. WEIN, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:518, 1927.

1. Operation is indicated in an uncomplicated frontal sinus suppuration in a medium or large sinus.

2. In a roomy sinus, it is without danger.

3. It is not necessary to remove all the mucosa. Good drainage with aeration is all that is necessary.

4. Anterior posterior and lateral pictures are desirable.

5. Resection of the upper foreward part of the septum is recommended as simplifying the operation.

IS OZENA CONNECTED WITH THE VEGETATIVE NERVOUS SYSTEM? BIRKHOLZ, Ztschr. f. Hals-, Nasen-u. Ohrenh. **18**:522, 1927.

The author presents anatomic (sympathetic nerve supply to the nose) and pharmacologic (the failure of sympathetic stimulants and vasodilators to show effect on the nose in ozena) arguments to show that ozena is a trophoneurotic condition. He also gives other theoretical arguments.

THE PATHOLOGY OF PERTUSSIS. GOLDMAN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:590, 1927.

As the result of careful investigation of a number of cases during an epidemic, the author believes that paranasal sinuses, especially the antrums and ethmoids, are the principal seat of infection. He calls attention to the symptomatology—first serous, later mucoid, nasal discharge. He believes that this dense glutinous discharge flowing down the nasopharynx starts the cough. The whoop, he thinks, is due to spasmodic contraction of the glottis following maximal pumping out of the air of the thorax by the prolonged cough, which brings about marked negative intrathoracic pressure, causing a powerful inspiration, and also aspiration of secretion, which secondarily infects the bronchi.

He says further that chronic paranasal sinus disease is the cause of the recurrence of pertussis.

He advises as treatment during the acute stage, rest in bed, diaphoresis, inhalations, suction and heliotherapy and heat for the head; later, irrigation of the affected sinuses, especially in stubborn cases.

LEWY, Chicago.

Miscellaneous

(From the Proceedings of the German Otolaryngologic Congress)

THE CAPILLARY BLOOD SUPPLY OF THE MUCOSA OF THE AIRWAYS AND ITS PHYSIOLOGIC AND PATHOLOGIC SIGNIFICANCE. STERNBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:593, 1927.

Microscopic examination of sections do not show which capillaries are functioning, i. e., those which are filled with blood, and those which are closed. Krogh has shown that in resting muscle the blood circulates through relatively few, while in active muscle many more function.

Normally, the mucosa of the airways is pale red to red, except that over the true cords, that of the accessory sinuses and tympanic cavity is pale yellow. In the absence of nasal respiration (tracheotomy), the nasal mucosa becomes blue-gray. The deeper one progresses into the lungs, the smaller are the bronchi, and the less is the subepithelial capillary injection. It is evident that the air stream stimulates the superficial blood supply. The author does not believe that inspired air is warmed much in passing the relatively small surface within the nose, but says that it is warmed in the alveoli of the lungs. He believes that the increased capillary injection of the nasal mucosa protects the mucosa itself against too much cooling, and quotes an analogous situation in the skin. He believes the same is true of the larynx, trachea and larger bronchi, the superficial blood supply gradually diminishing in the smaller bronchi as the necessity for defense against cold air diminishes.

The reaction to the Muck epinephrine-probe test (spotty reddening of the anterior end turbinate previously paled by epinephrine on irritation with a probe) occurs regardless of the contraction of the deeper lying erectile tissue, and is therefore due to the subepithelial vessels; this idea is further supported by the fact that the blue-gray color of the nasal mucosa so often seen in vasomotor rhinitis and asthma is present in spite of swelling of the underlying network of vessels. In these patients, after epinephrinization, irritation with the probe induces further contraction of the superficial capillaries; and this is evidenced by the local pallor remaining even after the effect of epinephrine has worn off and the rest of the mucosa has become relatively red.

The author used injections of India ink in his investigations, and thinks this method may be used to further the study of the blood supply in health and disease, and that it may lead to results in the study of "catching cold" and of the physiology of the labyrinth.

THE CHOICE OF ANESTHETIC. STUPKA, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:598, 1927.

Great progress has been made in the local anesthesia used in surgical procedures on the ear and the upper air passages. Anxiety, fear, etc., and sometimes the sensing of the patient that all is not going right must be considered, as well as the prevention of physical pain, which is not always entirely successful. Some of the newer, safe general anesthetic agents may make one reconsider the advisability of their use for patients and conditions not so well adapted for local anesthesia. A more definite classification of nervous disturbances should lead to a better choice. In neurasthenic persons of the exogenous type (Kraepelin, Gaupp) of moderate degree, local anesthesia is useful, supported by preparation with bromides, etc., the day before and morphine with scopolamine if necessary, the day of operation. Patients with severe conditions should be given a general anesthetic, and the others, if operation is not urgent, should receive preparatory treatment of rest, diet and sedatives.

Persons with endogenous disease of the nervous system, on a degenerative background, and psychopathic and neuropathic persons with abnormally high sensibility should not be operated on under a local anesthetic; on the other hand, persons

with organic diseases of the brain and nerves may be. Children in general should be judged by their age, nervous habits and their amenability to control. For short operations, "rauschanesthesia" is recommended. Tonsils and adenoids may be removed under ethyl chloride rausch, and in Neumann's clinic, a chloroform-ether mixture is used for this purpose. Anesthesia is carried only to the analgesic stage. The author prefers straight ether for various minor operations, and in very young children who cannot assist by spitting, etc., it is not harmful to induce anesthesia to the stage of unconsciousness.

He recommends highly the use of a suction apparatus as a safety measure against aspiration in ether anesthesia. Nonurgent operations should not be performed too soon after inflammatory processes, nor during epidemics of such (influenza). The mouth should be clean, infected teeth removed and antiseptic pastiles used before operation. Calcium preparations are said to diminish bleeding and the tendency to infections of the lungs; and injections of lobelin, 1 cc. are highly recommended for postanesthetic vomiting. Another precaution is warming the ether before administration.

The combination of local and general anesthesia may also be used thus greatly shortening the time required for general anesthesia.

The author emphasizes the danger of a patient's seeing or hearing something unfavorable during the operation, for instance unforeseen hemorrhage from a sinus, which may frighten him into a traumatic neurosis or cause him to discredit his physician.

A number of drugs are mentioned as useful for combined local and general anesthesia; incidentally, the use of cologne is suggested to obviate for children the disagreeable odor of ether.

Intracranial complications lend themselves well to local anesthesia, and in these ether is harmful to the already damaged brain tissue. Even meningitis may be so handled if the patient has a cloudy sensorium and is not too hypersensitive.

LEWY, Chicago.

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

C. J. IMPERATORI, ABSTRACT EDITOR

Fiftieth Annual Congress, Washington, D. C., May 1-3, 1928

D. BRYSON DELAVAN, M.D., *President, in the Chair*

PRESIDENTIAL ADDRESS. By DR. D. BRYSON DELAVAN, New York.

Published in full in this issue (p. 300).

DISCUSSION

DR. GEORGE M. COATES (Secretary), Philadelphia: I have been asked to announce that the President and Fellows of the American Laryngological Association appreciate the great courtesy that the American Otological Society has paid us by adjourning their meeting as a tribute to Dr. Bryson Delavan, our President.

DR. MAX A. GOLDSTEIN, St. Louis: Mr. President, as President of the American Otological Society, I have the honor to announce to you that the American Otological Society, your older sister in this worthy scientific activity in medical science, had adjourned to meet with you as a body to do honor to you, sir, on the occasion of this splendid semicentennial celebration.

PRESIDENT DELAVAN: Mr. President, I appreciate this compliment to the association more deeply than I can express. I also appreciate the attention to myself. I thank, you, sir, and your association.

Gentlemen, I declare the scientific session open and call for the reading of the first paper of the morning.

WHAT IS THE RELATION OF THE THYROID GLAND TO THE LARYNX? By DR. WILLIAM V. MULLIN, Cleveland.

Enlargements of one or both lobes of the thyroid may cause distortions of the larynx, and may produce, of course, the so-called saber trachea. It may push the larynx upward or pull it downward, or to one side, but this has practically no effect on voice.

Simple adenoma of the thyroid gland has absolutely no effect on the larynx or on the voice, unless we have the so-called enlargements or finger-like projections of one lobe into the pharynx, behind the larynx, and sometimes we have seen them passing into the pharynx and dropping down over into the top of the larynx.

Individuals with hyperthyroidism do have hoarseness and a weak voice, but this is due to a condition in the laryngeal muscles and in the extrinsic muscles that is likened to the tremor in the hands and to the muscular weakness all through the body.

I believe that the recurrent laryngeal nerve bears a far more intimate relation to the esophagus than it does to either the larynx or the trachea.

It seems that an enlargement of the thyroid gland, no matter how large, whether it extends backward or down, as a substernal thyroid, or intrathoracic thyroid, does not exert sufficient direct pressure on the recurrent laryngeal nerve in preoperative conditions to cause paralysis. I have not seen one case of laryngeal paralysis that could not be accounted for by some condition in the mediastinum, and not one case has been due to enlargement of the thyroid gland. After an operation has been performed, however, one sees a great deal of disturbance.

We are coming now to believe that when we find a laryngeal paralysis preoperatively, and particularly a bilateral abductor paralysis, we should look on that thyroid gland as being malignant. Thus, a bilateral abductor paralysis associated with the thyroid gland which has not been operated on, in nearly all instances, indicates a malignant condition of the gland.

On examining a considerable number of malignant thyroid glands, over 200, we have not found the larynx involved secondarily in any of them.

DISCUSSION

DR. JOHN F. BARNHILL, Indianapolis: It has been my experience that the aberrant goiter causes more laryngeal difficulty than ordinarily results from a goiter in the regular position. The aberrant goiter, of course, in its early stage, that is, in infancy, gives the greatest difficulty, and I think that as laryngologists, we should know that a considerable number of infants die during the first hours following birth from the so-called "circular" or misplaced goiter, which really chokes the child to death. I have had the privilege of making a number of postmortem examinations on children who have died from this cause.

The other aberrant goiter, sometimes overlooked, that gives rise to a great deal of difficulty is the goiter that does not descend from its original location in the tongue to its normal place on the neck — the goiter that lies in the base of the tongue and gives rise to great difficulty from hemorrhage and difficulties in deglutition and respiration.

I think the presenter is also correct in his statement that malignancy of the goiter does not often involve the larynx, except by direct invasion of the cartilage, neither does laryngeal cancer involve goiter very often.

DR. WILLIAM B. CHAMBERLIN, Cleveland: Dr. Mullin mentioned the fact that the relationship of the thyroid to the esophagus was much more intimate than it was to the trachea. On the other hand, is it not a noteworthy fact that dyspnea is more frequently associated with enlargement of the thyroid than dysphasia?

DR. GORDON B. NEW, Rochester, Minn.: The point that Dr. Chamberlin just brought up regarding the relationship of the esophagus to the recurrent laryngeal nerve was noted several years ago in experimental work on dogs. That is, if the nerve was pinched at a point low in the larynx, or was injured low in the larynx, the dogs had trouble in swallowing, and practically all died, while if the nerve was injured high above the fibers, as Dr. Mullin showed, passing through the esophagus, the dogs got along quite well and did not have trouble in swallowing.

I do not believe that traction, as Dr. Mullin brought out, can cause recurrent paralysis. We were unable to produce laryngeal paralysis in animals by long continued traction of the recurrent laryngeal nerve.

It has been my experience that in at least 95 per cent of injuries to the recurrent laryngeal nerve, the vocal cord was in the median line or abductor position. If the nerve has been permanently injured, the cord stays in that position. In the smaller percentage, approximately 5 per cent, in which the cord is in the cadaveric position, the cord will pull to the median line in from three to four months.

We used to hear that the patient was often hoarse after the operation, but as his voice improved he had trouble in breathing. In that case, the injury to the nerve was permanent, the cord pulled to the median line and the so--called median line abductor position resulted.

SIR ST. CLAIR THOMSON, London: I can recollect only a few occasions on which I was consulted by patients because of change of voice in thyroid disease, and I have rather patted myself on the back for discovering that this was due to early myxedema.

We discussed the subject of recurrent paralysis at Edinburgh last summer, and it was interesting to hear what a Swiss colleague had to say. I do not give his name, in case I do not quote him correctly, but he bore out what has been mentioned; it is the opinion in Switzerland that preoperative paralysis, associated with enlargement of the thyroid gland, is so suggestive that it is almost pathognomonic of malignant disease.

That is not what we find in our records in England. There are cases on record of paralysis existing beforehand and disappearing after a successful operation on the goiter.

With regard to the subject of operative paralysis, I have had occasion to see cases of goiter that were referred to me soon after the operation, in which the cords were mobile, and yet after a few weeks (or it may have been a month or two), paresis, perhaps a complete paralysis, occurred in one cord. On investigation, it seemed to me that it occurred in those cases which did not follow an entirely aseptic course — cases in which there was perhaps prolonged drainage and some suppuration, so that the paralysis occurred from this contraction secondary to sepsis.

I think that should make laryngologists, to whom these cases are referred, a little diffident in criticizing our surgical colleagues and condemning them as having cut the nerve, because this palsy might occur, if this thesis is correct, through no fault of theirs. In other words, we should "gently scan our brother man."

DR. MULLIN: Replying to Dr. Chamberlin, the relation of the thyroid gland to the recurrent nerve has a more intimate relation with the esophagus than with the trachea, but I do not believe that the enlargement of the thyroid may produce more disturbance of the esophagus than of the trachea.

Dr. New referred to what I said about traction. I did not mean to imply, Dr. New, that traction would produce the paralysis, but that traction would pull the nerve out of its bed, and in lifting up the gland would make it easy for the surgeon or the assistant, especially with severe hemorrhage, to grab some branch of this nerve.

I appreciate what Sir St. Clair Thomson has said.

A NOT PLEASING NOSE. By DR. VILRAY P. BLAIR, St. Louis.

In a selected group of people, none was encouraged to have an operation performed on his nose. In most cases, the patient is discouraged considerably before we consent to operate, and on the whole, we refuse at least two people to every one that is treated. But there are many people, and you have all seen them, I am sure, who, because of some real or fancied facial disfigurement (and often it is the nose), are obsessed with the idea that in their contact with their fellows attention is focussed on the nose and not on them or what they are endeavoring to do and it is an absolute handicap to these people in their course through life. That is my excuse for taking up this subject.

The types of noses that we deal with are congenital, developmental and those due to injuries which have not been corrected. In a general way, they include the flat bridge, the spread bridge, and in these days of the introduction of syphilis into parlor conversation, they are looked on with suspicion by the observer and resentment by the victim. The retracted type gives the impression to the victim of a feeling of insignificance. There are a number of different kinds, and when one classifies all that one knows about, other kinds will be seen.

FOREIGN BODIES IN THE STOMACH AND INTESTINES. By DR. M. C. MYERSON, New York.

To be published in full in the ARCHIVES.

DISCUSSION

DR. CHEVALIER JACKSON, Philadelphia: The presentation of Dr. Myerson represents the practically unanimous views of the esophagoscopists — those of us who see most of these cases and have to reach a decision as to what is to be done.

As to the question of gastroscopy, it arises in the case of every infant who is discovered to have a foreign body in his stomach. The question is raised, shall we perform a gastroscopy or not? We used to think it better to wait. We encountered a number of cases in which we were sorry we did wait. Our rule now is not to wait very long. We used to wait a month, now we wait only a

few days, or a week, to determine whether or not the foreign body is going to pass out of the stomach. The question as to whether or not it will be relatively safe to pass through the intestinal canal must be decided immediately. If it is a long needle, pin or an open safety-pin, we usually perform a gastroscopy at once. One of the most important points to consider is whether it will make the turn of the duodenum. That is the most dangerous point for two reasons: First, the sharp turn is likely to arrest the foreign body, especially if it is an open safety-pin. The other reason is that if arrested and operation is required, the duodenum is one region in the abdomen where the surgeon hesitates to operate for foreign body, especially on an infant.

Because of his study of the physiology of the intestinal canal, the roentgenologist is, in my opinion, the best man to decide in a given case, whether or not we had better perform a gastroscopy.

Any foreign body opaque to the x-ray, that has gone down through the mouth and esophagus into the stomach, can be removed by the same route from the stomach by gastroscopy. Gastroscopy, for its successful and prompt performance, requires the use of the double plane fluoroscope; without it, it requires a long time with the gastroscope before the foreign body will be found, unless it is long, like a stomach tube. Therefore, for quick localization, the x-ray is required. If one wishes to turn a safety-pin, to do an endogastric version, or if one wishes to close the pin, the use of the double plane fluoroscope is required. It is dangerous to go into the stomach with a gastroscope and forceps and try to deal with a foreign body, working only in one plane. You may think you are working right at the foreign body, and you may be 2 or 3 inches (5 or 7 cm.) posterior to it.

INTRINSIC CANCER OF THE LARYNX, OPERATED ON BY LARYNGO-FISSURE. By SIR ST. CLAIR THOMSON.

Fissure of the larynx, or splitting the thyroid cartilage, or thyrotomy is a term that should be employed only to indicate the route of approach. I also employ this route for a simple exploratory examination of the larynx. We may continue with the term "laryngo-fissure" in this sense; but students will start with mistaken ideas if they think it still means simply "splitting the thyroid cartilage and taking out a vocal cord." The operation I practice means excising a mass which contains in its center the whole cord, and includes the anterior commissure in front, part of the arytenoid behind, the ventricular band above and the subglottic area below. This excision goes deeply enough to include the perichondrium lining the thyroid cartilage and, occasionally, that on the inner side of the cricoid ring. As the greater part of one thyroid ala is also excised (to obtain freer access and secure better healing), it will be seen that a so-called "fissure" really means a partial laryngectomy.

Operations for malignant disease should be designed to meet variable conditions, the main object being to obtain a full face view of the whole extent of the disease so as to remove it, with a satisfactory margin of healthy tissue in all directions, while securing the safety of the patient and avoiding unnecessary mutilation.

Seventy cases are reported with an age distribution of from 30 to 80 years. There were seven women in this group. The first patient was operated on twenty-eight years ago. It is well to note that of the seven women, five remain alive without any recurrence, the longest time following operation in this group of women being eleven and one-half years. Among the men, the longest time following the operation is nineteen years. Of the seventy cases, there were eleven recurrences, eight of which recurred within the first year. There were eleven deaths from local recurrences. There were three patients who died within a few days following the operation.

DISCUSSION

DR. JACKSON: To say that a laryngofissure has been performed, does not mean that a cancer has been adequately excised. Laryngofissure, as brought out by Sir St. Clair Thomson, is a means toward the end, and not the end itself.

In witnessing his operation, it was striking to have presented to me on a piece of gauze, the single mass of tissue removed. It was a desert of normal tissue with an oasis of cancer in the center. Every surgeon and every medical student knows that it is fundamental in the removal of cancer to excise that wide area of surrounding normal tissue; otherwise the operation is almost doomed to fail. What struck me most forcibly in seeing Sir St. Clair Thomson operate was the access he obtained by the excision of the wing of the thyroid cartilage. The great difficulty in spreading apart the alae of the thyroid cartilage from a central incision, of course, is known to all of us. We have to work down, in and around to make the excision. When Sir St. Clair Thomson's technic is followed, one has the advantage of approaching from the side. In making his final incision, when the ala of the thyroid cartilage was out of the way, he made the final extirpation laterally. His approach was sidewise, although a central skin incision was used. It seems to me that this is a great factor in the excellent results that he has obtained, because it facilitated adequate removal.

One other point, I feel, deserves emphasis, because it concerns something that is neglected in this country. Sir St. Clair Thomson said that the public should be educated. The medical profession in this country recognizes the fact that hoarseness may mean cancer, but I am sure that the general public does not recognize the frequency with which not only chronic but intermittent hoarseness may be the first sign of cancer of the larynx. I think the public should be educated to the fact that when a man is hoarse, even though it is an intermittent hoarseness, his larynx should be examined and cancer excluded, and until this fact is recognized, we shall continue to see laryngeal cancer in an advanced stage. Most of the cases of cancer that come to our clinic are extrinsic by extension.

DR. BARNHILL: So far as I can see, there is no reason for removing a whole larynx in the beginning. There is every reason for removing the cancerous growth widely. I have never seen one removed as widely as these pieces would show and I certainly have never performed such an extensive excision, but I am glad to learn, as Dr. Jackson confirmed, that it can be done more easily by removing the cartilage.

DR. JOHN E. MACKENTY, New York: I have learned several things about Dr. Thomson's operation today. The operation amounts to almost a hemilaryngectomy. It is an extensive intrinsic operation, and it is the logical conclusion of the method which is known as laryngofissure. He carries the removal to a point short of closing the larynx, leaving space through which the patient may carry on the functions of breathing and speaking.

The subglottic extension is always greater than it appears from above, and that is the reason one never knows how extensive the disease is until the larynx is opened or removed.

I agree with the doctor in his conclusions about women. I have had three women patients under 27 years of age with cancer, two 26 and one 20. It would seem from my own personal experience that cancer of the larynx is more common early in life in women than it is in the later decades.

I think that eleven recurrences after seventy intralaryngeal operations is an excellent showing for a conservative operation, but it would be a poor showing in total laryngectomy, for the same type of case.

I should like to ask another question. How does Dr. Thomson keep the secretion out of the lung after operation, and supposing a slight hemorrhage occurs, how does he keep the blood from getting into the lung? Does he perform a preliminary tracheotomy or does he have some means by which the lung is blocked off for the danger period?

DR. PERRY GOLDSMITH, Toronto, Ont.: I should like to ask Sir St. Clair Thomson a question as to biopsy before operation. There are many pathologists, but there are few people familiar enough with the examination of these neoplasms to give an opinion on borderline cases.

SIR ST. CLAIR THOMSON: As Dr. Chevalier Jackson has said, I am glad to have insisted that a fissure is only the route, and that, as Dr. MacKenty says, it is really a partial laryngectomy.

We all know that hemilaryngectomies, performed in the old way, were more fatal than complete laryngectomies, and I hope that this more full removal from an endoscopic point of attack, will supplant the former, and generally dangerous and useless, hemilaryngectomy.

This brings one down to choosing between this extensive partial laryngectomy and a complete laryngectomy.

The removal of the ala is a point on which I continue to be well satisfied. The a priori suggestion is that the larynx may fall in. The old a priori idea, when we did resection of the septum, was that the nose would collapse, but the nose does not fall down. If we have the thyroid ala on each side like that (illustrating on blackboard), we must remember that here at the back we have the fixed arytenoid and in front the inner angle of the thyroid. If all one side is taken away, we still have the perichondrium quite taut from arytenoid to angle of thyroid, and there is no mechanical reason why that side should fall in. The perichondrium here forms tough fibrous tissue. We all know that cartilage in the human body is never restored, but in its place, fibrous tissue is thrown out by the perichondrium. This, stretching between those two fixed points, prevents any collapse.

In reply to Dr. Barnhill, I would say that I remove the ala for the following reasons: Access is easier. Instead of squinting through a narrow chink, a complete side is taken away so that the area can be viewed more completely. The bleeding is checked easier. A smoother surface is obtained for healing, because the inside of the perichondrium is a better surface for granulations to creep along than a bare surface of cartilage. We all know how long a bare surface of cartilage takes to cover over, and how likely it is that necrosis may occur. This method leaves a more vascular, smooth, healing surface.

I am also under the impression that the voice is much better when this method is used, and I am convinced that a larger glottis is secured. I also fancy, though it is not absolutely certain, that there are fewer granulations when the thyroid ala is taken away.

We should all agree, I think, on teaching the public with regard to hoarseness. The way I put it to students and family practitioners is to say that no human being, child or adult, should be continuously hoarse for more than three weeks without getting an expert opinion concerning the cause.

With regard to general care, first of all, I operate considerably under local anesthesia, namely, procaine hydrochloride, in addition, in our country, we also anesthetize the patient lightly with chloroform. We do not use ether for this operation, although ether is now the popular anesthetic. We use chloroform, because it does not irritate and just enough can be given to benumb the patient's sensibility. Before he leaves the table, his consciousness has returned. The patient is placed on the operating table in a sitting position, and we see that he is able to cough and spit. He is put back to bed in a sitting position, so that he can easily spit.

I perform a tracheotomy in all cases, and leave the tube in till the same evening. There has never been any difficulty with swallowing. That is why I try not to take away too much of the arytenoid.

These patients are operated on at about 9 o'clock in the morning and they are given water freely to drink beforehand. By evening they have touched nothing for seven hours. When I call at the hospital in the evening, the first thing I do is to give the patients some sips of sterilized water and, with rare exceptions, they swallow it well. With many of them any difficulty is mental; it is fear. They are encouraged to swallow, putting their chins down at each motion of swallowing, which makes it go better. If that swallowing goes well, the tracheotomy tube is taken out and is never replaced. The patients only have the tube for the first seven hours, which, in my experience, covers the danger period for hemorrhage.

All through the operation and after-treatment, the windows are kept open freely. The patient has water through the first night, and the next day we begin ordering food. He is out of bed always within twenty-four hours, sitting up, and, in many cases, the patient goes out for a walk at the end of three or four days. In many instances, they have traveled from London to Birmingham, Manchester, Glasgow

or Edinburgh, often an eight hours' railway run, at the end of ten days. I have never had a patient in the hospital more than three weeks.

There is no external dressing. A piece of gauze covers the wound which heals up like a shaving cut. There is sometimes a little sinus left for the first fortnight where the tracheotomy tube was.

The patients are encouraged to talk at the end of three weeks. I used to keep them absolutely silent for a month or six weeks until Dr. Jackson pointed out that the sooner one allowed a little functional use, the better. I then realized that my patients with the poorest voices were women, because the women were the most obedient and maintained silence so strictly that they often remained silent for six weeks. As a result they had the poorest voices, while the drunken old boozers, who would not stop talking and began to swear at the nurses early with one vocal cord, developed good voices. These are the compensations of virtue!

It is interesting to me to hear Dr. MacKenty say that women get cancer earlier in life than men. That is an interesting study, and those who make a special study of cancer in general might bear the point in mind.

As to the indications, it is rather difficult to lay down the limits, and one dreads being quoted incorrectly, but I would say that an intrinsic cancer limited to a vocal cord which is mobile, with both the anterior and the posterior commissuras free, is the most suitable case of all for operation by the laryngofissure route. Every patient with this condition ought, in the first instance, to have the operation I have described. If the cancer has extended to the arytenoid, I would not touch it with this method. If it has extended to the anterior commissure, I might risk it. If it has extended into the subglottic region, it is a little doubtful.

Biopsy is a difficult subject. If one could rely on the biopsy, it would be well, but in the first case I mentioned, in which an embedded growth was present, one could hardly get a sufficient piece on which to expect a pathologist to commit himself. If the pathologist gives a positive report, all is well; but if he gives a negative report, what is to happen? There is the possible fear, though I do not believe in it myself, that the traumatism stimulates the growth of cancer. I do not think that happens because I have watched cases in which I have performed operation two or three months after a biopsy, and they have been among my successful cases.

I do not think that there is as much fear of stirring up the disease as that the patient is lulled into a false security, and the surface of the cord is so damaged that a clinical opinion can no longer be formed. When the growth is projecting distinctly, or when there is doubt as to whether a soft-looking tissue is tuberculous (which is the disease most often confused with cancer), tuberculosis in middle aged and elderly persons without any symptoms in the chest or elsewhere being misleading — a good bit of tissue can be punched out of a soft tuberculous cord, and it is of some help. We are a more patient people in England than you are, and my patients are willing to come and be inspected once a month for three months. If there is a suggestion of tuberculosis, I send the patients to a sanitarium. I have watched patients for six months, without a biopsy, before I could make up my mind, and I have operated on them successfully. If the condition is malignant, the disease develops slowly and steadily, while the larynx is not improved when the use of tobacco or alcohol is given up or when teeth are extracted or tonsils removed. If the patient is tuberculous, the symptoms generally become evident within two or three months. So the biopsy is a rather difficult point to explain in a short discussion, but I think it is all the more urgent that we should teach our students that they must train themselves to see these things with a clinical eye; in fact, that they follow the catch phrase of one of the British statesmen, namely, "wait and see."

SEVERAL UNUSUAL CASES OF CANCER OF THE LARYNX. By DR. JOHN E. MACKENTY.

Squamous Cell Carcinoma of the Larynx.—The first patient had syphilis and had been hoarse for five years, because of a papilloma of the vocal cord. Radium

had been applied some time before the operation and the skin and subcutaneous tissues in front of the neck were badly damaged. The laryngeal growth had not been stayed in its progress. The patient suffered from toxemia caused by the radium plus the cancer. There was also an antecedent chronic laryngeal pathologic condition. Biopsy showed that there was a squamous carcinoma. Owing to the devitalization of the tissues of the neck, an esophageal fistula resulted following operation, and an artificial esophagus was devised. This took care of the saliva and permitted the patient to feed himself by mouth.

Intrinsic Squamous Cell Carcinoma of the Larynx.—The second patient gave a history of hoarseness and at times aphonia for eight years previous to the operation. Several pictures were taken during the period of observation. Tuberculosis and syphilis were excluded. Biopsy was advised some time before the operation, but was refused by the patient. Biopsy done on June 25, 1927, showed a chronic inflammatory lesion. About three months later, the larynx presented all the typical appearances of a fulminating cancer. The second biopsy showed squamous cell carcinoma. Operation was performed on Dec. 21, 1927, and was followed by osteoperiostitis of the right metatarsal bone. The patient had an uneventful recovery insofar as the laryngeal operation was concerned, and the jaw healed three months later.

Tuberculosis and Squamous Cell Carcinoma of the Larynx.—The third patient had tuberculosis with a sputum positive for the tuberculosis bacillus. He also had chronic nephritis and was a habitual user of morphine and heroin. He suffered from chronic hoarseness for several years. A clinical diagnosis of carcinoma of the larynx was made, and a biopsy was performed, a tentative diagnosis of tuberculosis being made. A month later, the Wassermann reaction was 2 plus, and *Spirochaeta pallida* was found in a small pustule in the patient's larynx. Antisyphilitic treatment was instituted for some months. Another biopsy was performed in October, 1927, and showed squamous cell epithelioma with tuberculosis in the deeper layers.

Tuberculosis, cancer and possibly syphilis were present in this larynx. There had been an antecedent pathologic process in the larynx for years as shown by hoarseness. Pain and dysphagia were present from the onset. Tuberculosis was probably present in the perichondrium. In this patient, just as in another patient in this series, the first biopsy was negative, the second was positive, but by that time, the clinical evidence was unmistakable.

Intrinsic Basal Cell Epithelioma of the Larynx.—The fourth patient was a girl, aged 20, who had sinusitis since childhood, enlarged glands of the neck and hoarseness for the past three years. Examination on June 29, 1927, showed chronic pansinusitis, infected tonsils and a neoplasm on the anterior half of the left vocal cord. Biopsy showed that the growth was a papillary cancer. High voltage x-ray therapy was used for three months. Operation was performed on Oct. 22, 1927, totally removing the larynx. Microscopic examination of the growth showed that it was a basal cell epithelioma. This is the youngest patient seen, and in spite of the apparent microscopic benignity of the growth, it grew with amazing rapidity and clinically seemed to be highly malignant.

A Two-Stage Laryngectomy. By Dr. Gordon B. New, Rochester, Minn.

If it becomes necessary to remove the larynx because of malignant growth, it is of first importance to remove the growth completely; the patient should be guarded against reaction, and the result should be a tracheal opening without the use of a tube. In the old two-stage operation of Crile, the patient usually was obliged to wear a tracheotomy tube. The one-stage operation of MacKenty, while overcoming this disadvantage and being technically more simple, requires a great deal of postoperative care from surgeon and nurses. The two-stage operation I am now using seems to combine the advantages of both of these operations without the disadvantages of constant close postoperative care, drainage tubes and frequent irrigations necessary in the one-stage operation and the occurrence of tracheal stricture in the old two-stage operation.

Thyrotomy and excision is performed without the removal of cartilage in cases of carcinoma involving the anterior two thirds of the vocal cords without fixation, the grade of malignancy, the age and general condition of the patient being taken into consideration. Laryngectomy is performed in the more advanced cases with fixation. During the three year period from 1925 to 1927 inclusive, 171 patients suffering from carcinoma of the larynx have been examined in the Mayo Clinic. Sixty-four of these were operated on. In seventeen instances, thyrotomy and excision was carried out, and in forty-two laryngectomy was necessary. This group includes certain extrinsic cases, but not the epiglottic or postcricoid cases in which lateral pharyngotomy, following the method of Trotter, might be performed. Exploratory operations were performed in five cases. Tissue obtained at biopsy was examined the day previous to the operation in all cases in which laryngectomy was to be performed.

There were three deaths following the first stage of the operation and none following the second stage in forty-two cases. One of the deaths was the result of infiltration with procaine of the region posterior to the larynx and trachea, a method used in the earlier cases in the second stage; as the patient could not swallow, he aspirated secretions and death occurred from bronchopneumonia after four days. This region is now infiltrated in the second stage after the trachea is cut across. Paravertebral block anesthesia as used for the second stage has been used in the Mayo Clinic in 876 cases (the majority of which have been bilateral) without injury to the pneumogastric nerves or other untoward results.

Operative Mortality of Carcinoma of the Larynx, 1925-1927

		Deaths
Thyrotomy and excision	17	0
Laryngectomy	42	3
Exploration	5	0
Total	64	3

The second patient who died was a man, aged 70, with cardiovascular disease; the systolic blood pressure was 240 and the diastolic 120. Death resulted from bronchopneumonia following the first stage. The third patient had a cough and slight bronchiectasis; the surgical risk was poor. Death occurred from bronchopneumonia after the first stage. If more care had been exercised in the selection of the cases, the last two deaths might have been prevented.

The points of interest in this method of laryngectomy are: (1) the use of local infiltration anesthesia for the first stage; (2) the median-line incision and splitting of the hyoid bone; (3) creating a barrier to infection by means of a clean wound; (4) opening the trachea later to infect the wound, and allow the patient to immunize himself and become accustomed to the opening; (5) performing the second stage of the operation under paravertebral anesthesia and infiltration of the pharynx, about eight days after the first stage; (6) complete primary closure of the wound of the neck without the usual drains or tubes, inserting the split tube below the tracheal opening; (7) applying gauze rolls laterally on the neck with pressure to eliminate the space previously occupied by the larynx and to support the pharynx, and (8) primary healing of the greater part of the wound of the neck and a tracheal opening without the use of a cannula in practically all cases.

DISCUSSION

DR. CLEMENT F. THEISEN, Albany, N. Y.: I want to ask Dr. New what he means by the patient "immunizing himself," and also to ask Dr. MacKenty how long after laryngectomy he has seen aspiration pneumonia.

I was particularly interested in Dr. MacKenty's case with regard to the premalignant stage. We have been able to watch a number of cases in which both tuberculosis and syphilis preceded the malignant condition in the larynx.

I am wondering just how rapidly malignancy follows the premalignant stage of either syphilis or tuberculosis.

SIR ST. CLAIR THOMSON: As I hinted in my paper, I am in entire agreement with Dr. MacKenty in his opposition to the use of radium for any operable condition connected with the larynx.

I should like to point out, too, what has been our experience in regard to the route of the spread of infection in some cases. When malignant disease starts on one cord, to one's surprise one finds that it has reached around to the other cord entirely in a subglottic region. It seems to descend from the first cord into the subglottic area and then cross the anterior commissure to the subglottic region on the opposite side, where it comes up onto the cord. The patient's voice may scarcely be altered.

Malignant disease in tuberculous subjects is now very well recognized, especially since it is now known that we are all, more or less, tuberculous.

The premalignant stage is interesting, and Dr. Chevalier Jackson's paper on it is a classic. There will always be some doubtful cases in these intriguing conditions of the larynx, but (not "if," because alas, it is a fait accompli) if after I had arrived at middle age I had a lesion of one cord which was thought to be not decidedly normal and yet not tuberculous, I would have my whole cord removed, and I would not bear any enmity toward the man who removed it if he afterward told me that examination by a pathologist had shown it to be normal. I think it is better to remove two cords and find them normal than to wait and let the condition of one cord become so far advanced that laryngectomy is necessary.

Dr. New has not quite persuaded me as to the cause of the death of his three patients. Surely people do not die from having an incision made over the laryngotracheal region, no matter how old and feeble and bronchitic they may be? Is he not perhaps withholding a criticism of local anesthesia and its possible dangers, particularly in the hands of those who are not expert? I ask that in pure innocence, because the use of local anesthesia, which I see is increasingly common here and on the continent of Europe, is still making little progress in England; and some people there claim that the deaths or the complications and secondary deaths due to local anesthesia have not been made public as they should be. I should like to ask Dr. New if he feels that the local anesthesia in these three cases, or in some of them, cannot be blamed for the unfortunate results.

DR. JACKSON: I was much pleased to hear Dr. MacKenty corroborate my observation made a good many years ago that there is antecedent nonmalignant disease in many of the cases of cancer.

About twenty years ago, I made the statement that one rarely saw cancer develop in a previously normal larynx. Sir Felix Semon questioned the statement and said that had not been his experience. I should like to ask Sir St. Clair Thomson to state his observations on that point. Another matter is the succession of cancer in benign growths. I reported to this association a number of such cases, to some of which Sir St. Clair Thomson did me the honor to refer, and one striking case has been observed since. One of the cases to which he referred was that of a nurse, who thirty years before she consulted me had had a papilloma in the larynx removed by Dr. Thomas R. French. He showed me an excellent drawing made at that time. When she came in to see me, she had a cancer of the larynx. It was verified by biopsy, and later, by her death from extensive malignant disease. It is inconceivable that that primary growth thirty years before could have been in any sense malignant.

The case that has since been observed I saw in consultation with Dr. Fielding O. Lewis. Dr. Lewis asked me to look at the larynx of a woman who had a condition that he thought was cancer. I looked at it, and I also thought it was cancer. I took a specimen, and it revealed squamous cell epithelioma. Eight years before, Dr. Sewall had removed a papilloma from the larynx and had submitted the growth to Dr. Ophüls. He could not find any evidence of cancer; yet when the patient came to us eight years later, she undoubtedly had cancer.

Dr. Lewis took out the entire larynx. She is alive and well and has a splendid buccal voice; but the question is, when did the succession of malignancy to benignancy occur? That such a transition does occur can no longer be logically disputed in view of such clinical evidence.

SIR ST. CLAIR THOMSON: I do not think that a frank papilloma or fibroma becomes malignant, but the manifestation of malignant disease is, in a certain number of cases, foreshadowed by years, sometimes, of some vocal defect.

The variety in the virulence of cancer infection is inexplicable, and when we know more about it I do not think it will be found to be a change from one condition to another. It is all the same, only that in some cases there is a longer gestation of the disease than in others.

DR. DELAVAN: Let me recall to you that Dr. J. Solis-Cohen many years ago operated on a patient with an extensive carcinoma of the larynx, the first in this country in which the trachea was brought out at the base of the neck and fastened to the walls of the incision. Dr. Solis-Cohen personally carried out the post-operative care of the patient until the danger period had been passed. The patient recovered, and in course of time developed a pharyngeal voice, the first and one of the best ever developed in this country. With this case, innovations were introduced, each one of supreme value, and each have been extensively followed since by all who have performed the most successful operations. When last heard of, this patient was still alive. Although minor improvements of value have been suggested by others, the fundamental principles of treatment practiced by Dr. Solis-Cohen are the basis of a large part of the improved statistics of the present day.

In the department of partial laryngectomy, as far as I can learn, a patient of mine, operated on by Dr. Farquhar Curtis thirty-one years ago (1897) and today in perfect health, shows the longest survival on record.

With regard to radium, the question of its value is by no means settled. While it may be true that ignorantly and improperly applied it may do harm, one must admit that the knowledge of its application in the larynx is still in its infancy. Forty years ago, the statistics of laryngectomy were uncompromisingly unfavorable. Von Bergmann of Berlin reported total extirpation in twenty cases; six of the patients died on the operating table and only three were apparently well at the end of two years. He also reported nineteen cases in which partial extirpation of the larynx was performed; five of the patients died on the operating table and only four were supposed to be well at the end of two years. The operative statistics of Sir St. Clair Thomson and Dr. MacKenty offer wonderful contrasts to those who early performed these operations. To them belongs the credit of having furthered the improvements which have brought about substantial betterment. If the surgical procedures on the larynx have so advanced, why may not radium treatment show similar improvement with more experience in its use? Already, through the use of radium in experienced hands, there has been a distinct and steady advance in the success attending its employment, as witnessed by the experiences reported from Scandinavia and Holland.

DR. MACKENTY: With regard to Dr. Theisen's question about aspiration pneumonia, I have seen only one case of aspiration pneumonia in our cases which number more than 200.

We prevent aspiration pneumonia by not allowing any blood to enter the lungs at the time of the operation, not even a drop, if we can help it. Then we prevent any secretion from the wound passing the barrier during convalescence.

I wanted to ask Dr. New whether they use the tracheal plug in their two-stage operation or whether they just put a loose tracheal cannula into the trachea.

I am glad to get a little corroboration concerning the use of radium. I am particularly interested in its action on the squamosa type of cancer. Here it has been most harmful. The radiumologists say: "Give us something less malignant, something like the basal-cell carcinoma to treat." Even in less malignant conditions they have failed.

I agree with Dr. Thomson that it is curious how the cancerous process will dip down under the commissure and involve the other side of the larynx. There must be some resistance about the tissues of the commissure which prevents crossing on the line of the cord.

I have seen malignant disease and active tuberculosis together in the same larynx only once before.

SIR ST. CLAIR THOMSON: I should like to make this clear. I have never seen cancer and tuberculosis combined in the same larynx. I have seen one condition follow the other. I only meant I have seen cancer of the larynx in tuberculous subjects, but I have never seen the two diseases present in the larynx at the same time.

DR. MACKENTY: The device to which Dr. Delavan referred is the artificial larynx, known to all laryngologists now, since it has been in use for four years. The artificial esophagus was the outcome of failure to obtain healing in the necks burned by radium. This device fills in the dehiscence in the esophagus, directs food and saliva into the stomach and relieves a miserable situation.

DR. NEW: Regarding the use of the word "immunization," it is not a good word, but I could not seem to find a better one. I open the trachea and allow the secretion from the neck to flow into the wound for four or five days. Thus, on removal of the larynx, the patient does not have the sharp reaction that sometimes occurs following laryngectomy.

With regard to the cause of death in these three cases, the first case was undoubtedly one of aspiration pneumonia. The anesthetist blocked the nerve posterior to the larynx at the first stage, so that the patient's pharyngeal secretion ran directly into his larynx and aspiration pneumonia resulted.

PRESENTATION OF THE DEROALDES MEDAL AND THE CASSELBERRY PRIZE.

DR. CHEVALIER JACKSON was presented with the De Roaldes Medal, he being the first fellow to receive this award for having contributed the greatest gift to laryngology.

THE EMBRYOLOGY AND NEUROHISTOLOGY OF SPHENOPALATINE GANGLION CONNECTIONS; A CONTRIBUTION TO THE STUDY OF OTALGIA. By DR. RALPH A. FENTON and DR. OLOF LARSELL, University of Oregon Medical School, Portland. (Casselberry Prize, American Laryngological Association, 1928; Read before the American Otological Society, Washington, D. C., May 1, 1928.)

The study of otalgia of sphenopalatine origin has been disappointing to Sluder, Müller, Terracol, Bertein and others, who have been handicapped by the difficulty of obtaining proper material. The anatomic pathway for these sensory impulses, erroneously ascribed to sympathetic fibers incapable of such function, was partially worked out in Ramsay Hunt's clinical studies of herpes oticus, 1907-1915, though he was unable to secure actual anatomic proofs.

Larsell and Fenton, accepting the sphenopalatine as a sympathetic ganglion with characteristic multipolar cells, point out its origin in comparative anatomy along the course of the palatine branch of the primitive seventh nerve. They also emphasize the fact that lower forms of life show numerous physiologically different sets of nerve fibers, readily traceable by size, myelin sheathing, etc., in the seventh, ninth and tenth cranial nerve trunks, and to a lesser degree in the fifth. Thus the fifth and seventh nerve trunks are much alike in lower vertebrates, while in mammals reapportionment of function leaves the fifth largely sensory and the seventh largely motor.

Although they point out that the sphenopalatine ganglion cells, sympathetic in character, receive small preganglionic fibers through the great superficial petrosal nerve, the authors demonstrate numerous large medullated fibers of the sensory type which run through the sphenopalatine and great superficial petrosal to reach

cells in the geniculate ganglion; these sensory fibers come from palatal and nasal distribution.

Confirming Ramsay Hunt's clinical studies and Rhinehart's work on the mouse, Larsell and Fenton have found in the human fetus a small facial branch distributed through the auricular branch of the vagus to the posterior part of the auditory meatus and tympanic membrane and to the cranial surface of the concha and skin over the mastoid. Its fibers rise from cells in the geniculate ganglion.

The great superficial petrosal nerve may be considered a mixed trunk, consisting of a white ramus communicans plus the palatine seventh. The geniculate ganglion, into which the cutaneous auricular branches of Rhinehart pass, is a sensory ganglion containing unipolar cells of the usual craniospinal sensory type. Large myelinated fibers from the palatine distribution are traced by the authors through the sphenopalatine ganglion without interruption into the great superficial petrosal nerve and then to cells of the geniculate ganglion.

Larsell and Fenton thus establish the conditions needed elsewhere in the body for reference of pain from a visceral region to a body surface. Irritation in the sphenopalatine region stimulates the palatine seventh fibers through the great superficial petrosal to cells in the geniculate ganglion; these cells are directly related to adjacent cells from the fibers of the cutaneous branch of the facial nerve, described by Rhinehart, transferring the pain impulse to the auricular and mastoid region supplied by this nerve.

Further work on the deep petrosal sensory tracts, carotid plexus, and vagus and glossopharyngeal connections is in progress and may throw some light on asthma, allergic and anginal phenomena.

DISCUSSION

J. Gordon Wilson approved of the validity of the anatomic demonstration. He gave extensive anatomic and clinical bibliography and presented nine detailed figures.

THE BIRTH OF LARYNGOLOGY. By DR. D. BRYSON DELAVAN.

The fiftieth anniversary of the American Laryngological Association commemorates a notable accomplishment in the history of modern medicine, worthy of recognition and of national pride, for the science of laryngology as well as that of gynecology is the outcome of American genius. Both originated in the United States. Founded in 1878, half a century ago, the American Laryngological Association was the first national society of laryngologists in the world, the next in order being the British association founded by Sir Morell Mackenzie in 1888. It has fluorished continuously since its inception and is today in the full tide of vigorous prosperity.

Thirty years before the birth of our association, the science of laryngology was founded by Horace Green of New York, whose discovery of the tolerance of the larynx to the presence of a foreign body opened a new era in the history of medicine, for on this basic physiologic principle depends in large measure all the achievements of modern laryngology. Antedating the invention of the laryngoscope by many years, Horace Green added to his discovery a knowledge of the diseases of the upper air passages and developed methods of treatment which enabled him to devote his entire attention to the subject, to the exclusion of the practice of other branches of medicine, in which nevertheless he was one of the authorities of his days. He thus inaugurated specialism in our art and was in fact its first votary, maintaining his position with such success that discriminating critics of the highest authority have insisted that under the influence of his teaching, laryngology would have lived and flourished even had the laryngoscope never been invented. Receiving the general plaudits of intelligent observers abroad, Dr. Green was unhesitatingly accorded the distinction of "Father of Laryngology."

While Green was announcing his discoveries on the physiology of the larynx and their application, Gurdon Buck of New York, the most famous general surgeon

of his time, in 1848 published his method for the relief of obstruction to breathing due to edema of the glottis, operating in the dark, aided only by his sense of touch. The value of his suggestion was at once recognized and received with widespread approval. Dr. Buck also successfully removed several laryngeal tumors and greatly improved the procedure of tracheotomy. His ideas gained for him both here and in Europe the title of "father of intralaryngeal surgery."

The work of Horace Green and Gurdon Buck directed general attention to the subject of the larynx, and was followed a few years later by the invention of the laryngoscope by Manuel Garcia. In 1858, Horace Green procured a laryngoscope and immediately introduced its use in the treatment of patients. He was the first in this country to appreciate its value and use it clinically.

The invention of the laryngoscpe ushered in the grand epoch in the history of laryngology. During the sixties, progress in this country was greatly retarded by the Civil War, but meanwhile the use of the laryngoscope had been introduced into Europe and its teaching carried on by men under whose masterly influence were trained the most distinguished teachers of the following generation, who in turn became the instructors of our pioneers, many of whom had spent a year or two abroad for the purpose of this special study. With the return of these well trained students in the early seventies, laryngology received the impetus which was thoroughly to establish it in the confidence and esteem of the public, and rapid advances were made.

The first of these was the publication, in 1872, of the pioneer work of Dr. J. Solis-Cohen of Philadelphia, a compendium of the knowledge of the day together with invaluable suggestions of his own. This was the first complete system published either in this country or in Europe.

Among the most active workers of the time was Dr. Clinton Wagner, who, in 1873, established in New York the first hospital in this country devoted exclusively to the study and treatment of diseases of the nose and throat, and inaugurated in it the first regularly organized courses of instruction for graduates in medicine.

Of still greater importance, he became the founder, in October, 1873, of the New York Laryngological Society, the first local association of its kind in the history of laryngology. The proper place which this society should have been given in history has unfortunately never been accorded it, for far from having led a short and insignificant life, it flourished from the time of its origination until it was merged into the Section of the Laryngology of the New York Academy of Medicine in 1886. This society was the arena in which was exploited the pioneer work of Wagner, Bosworth, Jarvis, Beverley Robinson, Lefferts, Sass, Lincoln, French, Asch and others of the brilliant group which composed its membership. Within it was originated the science of rhinology. Catlin, missionary to the Indians, years before, had first called attention to the evils of mouth breathing, a subject later emphasized by Clinton Wagner; but it was Bosworth who first directed attention authoritatively to the subject of nasal obstruction as influencing this condition. In 1881, his great work appeared in which the subject of rhinology was carefully presented. These and many other original contributions on subjects pertaining to laryngology and rhinology were in no small degree the result of the presentations and discussions held at the regular monthly meetings of this progenitor of the laryngological societies of the world.

Other early contributors to the development of rhinology in this country were Harrison Allen, John N. Mackenzie, Shurley, Bryan, Daly and Roe. The pioneer work of Bryan in the study of the anatomy and pathology of the nasal sinuses and the original and ingenious methods devised by Roe for the repair of deformities and injuries of the nasal framework long antedated similar work abroad. The same may be said of the work of Bosworth and Asch. Emil Mayer and Roe are on record as having been the first to advocate and practice submucous resection. To Roe has been accorded the title, "father of intranasal surgery."

Notable advance in the technic of the treatment of the throat was made in 1872 by Dr. Luis F. Sass of New York, who introduced the spray apparatus for the application of medicaments to the upper air passages, thus eliminating the

nasal douche, the postnasal syringe, the sponge probangs and laryngeal brushes employed up to that time.

During the eighties, notable contributions to laryngology were made by Joseph O'Dwyer of New York, whose most brilliant gift was his discovery of the tolerance of the larynx to the continued presence of a foreign body. In this, he supplemented Horace Green's great contribution to physiology and founded on it a series of ideas and practical accomplishments which have made him famous in the history of medicine. These include the affording of instant relief in cases of acute obstruction of the larynx, the cure of patients with chronic obstructions as never before possible; and, finally, the advancing of the basic idea from which has been developed the whole science of bronchoscopy for the removal of foreign bodies from the trachea and bronchi. O'Dwyer devised a tube of thin walls and large caliber through which the trachea and entrance to the bronchi were explored by means of the laryngoscope; the expulsion of the foreign body was aided by placing the patient on the back with his head and shoulders low—the expulsive efforts of coughing being aided by gravity in the dislodgement of the foreign body—all this was before the days of local anesthesia and the endoscopic light. The death of O'Dwyer prevented his further development of this idea.

Another notable contributor to laryngology was Dr. Thomas R. French, who many years ago devised a method for the photographing of the larynx which has never been equaled, and who demonstrated its practicability and usefulness for the study of the physiologic action of the larynx in vocalization and of its pathologic conditions. Many other contributions of value have been made by Dr. French, among the latest being valuable additions to our scientific knowledge of the pathologic changes of the tonsil.

To Dr. Robert Abbe, of New York, belongs the credit of having introduced the application of radium in the treatment of benign laryngeal growths.

The pioneer work of these great contributors and others who have succeeded them presents indisputable evidence that the fundamental principles of laryngology as it is known today were born of the genius of our fellow-countrymen. The record of American laryngology is preserved in the unbroken series of the fifty volumes which compose the "Transactions of the American Laryngological Association."

Looking Backward on American Otolaryngology in the World War.

By Harris P. Mosher, Boston, of the United States Army, Medical Corps.

In the beginning of the war, the Section of Otolaryngology was the busiest of all the sections of the surgical specialties. It always remained moderately active, but the Section of Plastic and Oral Surgery, now called maxillofacial surgery, and the Section of Neurological Surgery later received from overseas more of their special cases than the Section of Otolaryngology. The section of Neurological Surgery received by far the most cases, the majority of them being injuries of the peripheral nerves.

In the United States, the two great medical problems of the World War were the control of sputum-borne diseases and the detection and cure of the carrier. The bacteria responsible for sputum-borne diseases gain their entrance to the body in most instances through the tonsils or the accessory sinuses. Experience showed that the best method of curing a carrier was to remove the tonsils and the adenoid. The war showed also the supreme importance of accepting in the army only recruits who have a normal nose and throat.

The war showed, further, the importance not only of discovering carriers, but of segregating those who came in contact with patients. It brought out the significant fact that the hospital personnel often act as carriers. In dealing with air or sputum-borne diseases, the great value of isolation and the use of substantial gauze masks was proved. The probable efficiency of the routine spraying of the nose and throat with dichloramine-T was shown. The opinion finally prevailed that every recruit and every patient in a hospital should be looked on as a possible carrier.

In the United States, the dominant bacteria have been the pneumococcus and *Streptococcus hemolyticus,* the latter being by far the more important. The following fact has become more and more evident, namely, that assembling men in barracks, especially exposing them to cold, and drilling them to the point of overfatigue has the power to change harmless bacteria into virulent strains.

In operations on the head, America's greatest contribution has been in the surgical procedures on the jaw. Bridge splinting, the uses of bone grafts, the implantation of cartilage to give form to plastic flaps and the use of the skin graft folded over a plate of modeling compound greatly advanced maxillofacial surgery.

The fact that so many tonsillectomies were performed with so few deaths from any cause and only one death from tonsillar hemorrhage speaks well for the skill of the surgeons and the type of operation employed. The striking number of peritonsillar abscesses would seem to show that even a larger number of tonsillectomies could have been performed with advantage.

The influenza epidemic was a dramatic and deadly scourge, but the complications produced in the line of otolaryngology were not serious. It emphasized the fact, however, that a normal nose and throat is a great protection.

The otolaryngologists faithfully and enthusiastically used their specialty for the prevention of disease as well as for its alleviation. Their first aim was preventive surgery, and they largely realized it.

In looking backward over experience in the war, it is seen that some things could have been done better. Recording them may help another time, if there ever is another time. The following are some of them:

The draft examination should have been more thorough. Too many cases of chronically running ears were overlooked.

There could have been closer cooperation between the camp and the base hospital. There was no provision for an automatic interchange of special personnel.

Special instruments and supplies were late in getting to the hospitals.

Instruments, medical supplies and literature were late in getting overseas.

The hospitals at the port of embarkation should be thoroughly equipped and supplied with an ample staff. Soldiers destined for overseas service often arrived at the "port" seriously ill.

In the United States, the power to act as consultant should have been given earlier to the chiefs of the sections.

Each medical and surgical section should have a medical officer to act regularly as courier between the home base and the base of operations.

The Division of Surgery of the Head functioned most fully in this country. Its best work was in preparing men for overseas service and then in receiving them back for employment in its special forms of surgical procedures.

Specialization can easily be overdone, especially near the front where the majority of operations are performed on account of trauma.

The two sections of the Division of Surgery of the Head that were of most value at the front were the divisions maxillofacial surgery and ophthalmology.

Air and sputum-borne diseases were the outstanding medical problem of the war, and the problem still remains unsolved.

When it is discovered how to produce a general instead of a specialized immunity, preventive medicine, and army medicine should be chiefly preventive, will make one of its greatest advances.

When I left the army, I felt that my advice and that of my confreres might on occasion be of use in time of peace. I joined the Medical Reserve Corps. I confess that I have not been faithful to its duties. Nevertheless, I could have answered a question now and then had it been asked. I am curious to learn what has been the experience of the other senior men who are in this audience.

Finally, I recommend the use of the facilities of the American Board of Otolaryngology to the peace time Medical Corps of the Army. I have more than a suspicion that its services are needed.

DENTAL CARIES IN PARANASAL SINUS INFECTIONS. By DR. GORDON BERRY, Worcester, Mass.

Able and careful authorities have taught that 20 per cent of maxillary sinus infections are of dental origin. I believe that it would be a more acurate estimate to consider it from 60 to 80 per cent.

An analysis of 152 cases would seem to show that this is not overestimated.

The table shows the relationship between dental caries and paranasal diseases in 152 cases analyzed, showing percentages.

Relationship Between Dental Caries and Paranasal Disease

Of Dental Origin	None	Possible	Probably	Proved	Total
Acute maxillary.....	3	11	5	1	20
Chronic maxillary.....	4	21	18	16	59
Chronic ethmoidal.....	1	4	1	1	7
Nasal polypi.....	3	5	6	0	14
Total	11	41	30	18	100

Conclusions.—Though infections of the sinuses occur in the young, they are much more frequent in adults and in those in the prime of life. This is not a period when the anatomic nose is growing or changing and thus impeding sinus drainage, unless external traumatism is superimposed; the tonsils are offending no more or but little more than during adolescence; senile infirmity is not yet present; there should not have been any alteration in the sensitiveness to hay-fever and asthma; the patient is usually in fairly rugged health. These factors alone do not adequately explain the higher incidence of sinus infections. On the other hand, the teeth are decaying during this period. They are anatomically in close proximity. Advancing dental caries sooner or later reaches the pulp chamber, and the easiest path for an infection caught under the filling or crown is up through the dental canal to just beneath the antral floor.

I have relied on the x-ray film to show a dental abscess. My contention is that the relatively prompt discharge of this infection into the antrum stops the bony reabsorption looked for in the film; and that in this type of case, unless new methods for diagnosis become available, it is the rhinologist, working with the dentist and roentgenologist, who will determine whether harm is resulting.

A dead or dying tooth in close proximity to the antral floor, no matter how harmless appearing, is a threat. Recurring pains in the antrum on this side, an occasional seepage of pus from the middle meatus, small polypi in this region, even a chronic unilateral nasal congestion otherwise unexplained, should attract attention to the teeth or to a possible buried root. The finding and removal of such a nearby septic focus will clear many a stubborn nasal and paranasal infection. This is a reasonable claim and has the support of experience.

DISCUSSION

DR. FRANK R. SPENCER, Boulder, Colo.: I should like to ask Dr. Berry how he proceeds with this transilluminator.

DR. LEON E. WHITE, Boston: I have found that devitalized teeth, not necessarily abscessed teeth, are as a rule a more common source of infection than real abscessed teeth.

When an abscess does not form and yet infection takes place, it immediately gets into the system, and Dr. Berry considers that any tooth which is a potential source of infection, should be removed.

He also speaks of teeth which contain a great deal of filling. In looking over any patient who comes to me, when I suspect dental infection, I examine carefully the fillings in the teeth. If I see a molar that is heavily filled, I consider it a source of menace. If I see a crowned tooth, I consider it a source of menace.

DR. THOMAS E. CARMODY, Denver: The age of Dr. Berry's patient is somewhere around middle life, when not only abscessed teeth must be taken into consideration, but pyorrhetic teeth.

I have been using the transillumination that Dr. Berry has spoken of, for some time. The x-ray specialist knows the angles at which the roots come off the teeth, and he will give you much better service than the general x-ray man.

Any dead tooth is a threat, not only to the sinuses, but from the standpoint of focal infection, and that must be considered in many of these cases.

DR. WILLIAM B. CHAMBERLIN, Cleveland: Dr. Weston A. Price of Cleveland, as probably most of you know, is practically the father of x-ray investigation of the teeth. He took up this study in an early day, and built his own x-ray machine. In a conversation with him just a short time ago, he told me that he felt sure he could read 50 per cent of these dental films, but that he was unwilling to express a positive opinion about the other 50 per cent.

Dentists in Cleveland believe that practically all cases of antral disease are of dental origin, and that there is no other possible source of infection—a rather amusing position.

DR. BERRY: Dr. Spencer requested information about the transilluminator I use. It is the one made by the Cameron Instrument Company.

Dr. White spoke of the crowned tooth as a menace. He also said that the dentist was the wrong man to consult. The particular man whom I have found most serviceable (and Dr. Carmody kindly bore me out) is a dentist who does nothing but x-ray and diagnostic work.

FURTHER EXPERIENCE WITH THE FRONTAL-ETHMOSPHENOID OPERATION UNDER LOCAL ANESTHESIA, PRESENTING A NEW OSTEOPLASTIC FLAP. By DR. EDWARD CECIL SEWALL, San Francisco.

Published in full in the August issue of the ARCHIVES (p. 144).

DISCUSSION

DR. HILL HASTINGS, Los Angeles: I think that some of the radical procedures used in operations on the ethmoid sinus in the past have been thorough but not safe, and there have been many deaths that have never been reported. Sewall offers a procedure that I think is not only thorough, but safe. As to the operation, it is all that he says; it is bloodless, or almost bloodless. I was astounded to see everything better than I could in the mastoid operation. I was astounded at the perfect approach to the ethmoid and sphenoid. The ligation of the anterior ethmoidal artery is the crux of the situation.

DR. SPENCER: I saw Dr. Sewall demonstrate his operation two years ago. First, he had remarkable anesthesia. Second, there was apparently absolute freedom from hemorrhage. The method of ligation certainly does much to improve the operation as compared with most external operations on the sinuses. Third, I noted the thoroughness of the operation.

DR. GOLDSMITH: Dr. Sewall advocates opening of the ethmoidal labyrinth in its upper and posterior part under direct vision, and in that way carries out the better part of the cardinal principles on which good surgery is based.

I believe it is the operation of the future in cases of chronic frontal ethmoidal disease, but my fear is that those overzealous surgeons will tend to operate in cases in which cure might have been effected by simpler and more conservative measures. The operation should be restricted to the cases of chronic recurring infection of the frontal ethmoid sinuses that do not clear up even when heroic measures are used, and those in which the symptoms are sufficiently severe to demand a surgical approach.

DR. LEE M. HURD, New York: I am glad that the use of scopolamine morphine is getting to be a regular procedure. I advocated it for many years.

I have been performing an operation nearly like the one Dr. Sewall employs, except that I have not been so careful about hemostasis. I think that is well worth taking into consideration.

I can offer one suggestion as to something that I have been doing for a few years to maintain the opening of the frontal sinus. Take out the middle turbinate, peel off the mucous membrane, and when the frontal sphenoethmoid operation is completed, lay that mucous membrane down into the frontal orifice, letting it stay there without any packing.

DR. BERRY: I gathered from Dr. Sewall's paper that the mucous flap from the nose is attached by its pedicle and the blood supply is preserved, and from the pictures that the mucous flap is an isolated flap, and the blood supply is all from the skin side. I should like to have that point cleared up.

DR. CLYDE LYNCH, New Orleans: I had the privilege of seeing Dr. Sewall operate two years ago, and I thought that one of the most dramatic points in his operation was when he cut the nasal branch of the sphenopalatine artery and his patient gagged and bled. He announced during the procedure that the vessel had been cut, and proceeded to ligate it. After ligation, the bleeding stopped and the operation proceeded giving evidence that he had done what he said he would do.

Since viewing his work, I have ligated the anterior ethmoidal, the posterior ethmoidal and the nasal palatine branch from the sphenopalatine artery, and it has been a source of great comfort in the proceeding of the work for the pansinus operation.

DR. SEWALL: In answer to Dr. Berry, I would say that when this flap is cut, the chisel perforates the bone, and is followed by a knife; the mucous membrane lying on the inside of that bone is nasal mucosa and is not disturbed in its original location. It is simply cut and left on the inside of the bone and it receives its blood supply through the bone.

ETHMOIDITIS IN INFANTS AND YOUNG CHILDREN WITH ACCOMPANYING EYE AND ORBITAL COMPLICATIONS. By DR. CLEMENT F. THEISEN, Albany, N. Y.

To be published in full in the ARCHIVES.

DISCUSSION

DR. ROSS FAULKNER, New York: The subject of sinusitis in children always seems to be interesting.

I am much interested in orbital complications of sinusitis in children. Dr. Theisen has emphasized conservatism. Conservatism, to a certain extent, is a matter of age, regardless of experience.

About ten years ago, almost every case of orbital complication from a sinusitis in children that came into the hospital with which I am connected was regarded as a case for an emergency operation. The patient was operated on almost immediately. These patients would come in with exophthalmos and swollen lids, with a high temperature, and they would be put in the operating room within twenty-four hours.

I claim some credit for changing that order of things. Those cases are rarely urgent enough to necessitate operation. Like every other acute suppurative process, if there is time to wait until nature supplies some local immunity or promotes some barrier around the acute suppurative process, the operation can be performed with much more safety. By waiting a short period, this operation can be performed in a conservative way and with comparative safety.

An absolute fixation of the eyeball is perhaps more important than the exophthalmos. That will probably indicate that the ethmoids must be opened.

DR. SPENCER: Pediatricians now are apparently well educated regarding the importance of early recognition of sinus disease, and if we can educate the family physician we will have an opportunity to carry out Dr. Theisen's theories that these are not all surgical cases, and that conservative treatment may clear up the condition early. We all try to educate these physicians, but some campaign which will reach more of them, will do much more good.

OSTEOMYELITIS OF THE FRONTAL BONE; FOUR CASES PRESENTING PROBLEMS IN ETIOLOGY AND TREATMENT. By DR. VIRGINIUS DABNEY, Washington, D. C.

Osteomyelitis of the frontal bone following a radical surgical procedure is due not to the operation and its technic, but to long contact with undrained infective material. Another potent cause is the hemolytic bacteria, especially the streptococcus. Dr. Dabney presented four cases in detail.

DISCUSSION

DR. BLAIR: I think Dr. Dabney's record is unique in that all of his patients with this type of osteomyelitis recovered as a result of the treatment. The forty cases that MacKenty based his conclusions on in 1913 — forty-one, with one of his own — and six that I think can be added from the literature as far back as 1843 are probably associated with disease of the frontal sinus.

Statistics show that there are thirty-three articles published concerning those forty-six cases, and most of them are in favor of early radical operation. I think Mosher is the one who stands out most prominently as cautioning against that.

It is difficult to draw any conclusions from these few cases, but the facts as I make them out are that in those cases radical treatment was employed in thirty-seven cases; twenty-five of the patients died and twelve lived. Conservative treatment (and this was given in the older cases) was given to only nine patients; of the nine, three died and six are living.

From 1913 to 1925, of the patients given the radical treatment, eleven died and eight are living; of those given the conservative treatment, two died and four are living. Adding to that the six cases of our own, these are my conclusions: In fifty-six cases in which radical treatment was employed, 35 per cent of the patients are alive and 64 per cent dead; in twenty-one cases in which conservative treatment was employed, 61 per cent are alive and 38 per cent dead.

DR. CARMODY: I have seen five cases, and in these, one patient had a syphilitic taint, so that case should be eliminated. Three patients died and two recovered; one of those who recovered was the syphilitic patient, so that my death rate has been 75 per cent in the cases of pure osteomyelitis. All of them, except one, had chronic infections like those in the cases Dr. Dabney reported.

Directory of Ophthalmological and Otolaryngological Societies

INTERNATIONAL

FIRST INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS

President: Prof. E. Schmieglow, University of Copenhagen.
Place: Copenhagen, Denmark, Time: July 29-Aug. 1, 1928.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: William V. Mullin, Cleveland Clinic, Cleveland.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Portland, Ore. Time: July 8-12, 1929.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. C. J. Imperatori, 17 E. 38th St., New York.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Bryson Delavan, 40 E. 41st St., New York.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Hill Hastings, 1136 W. 6th St., Los Angeles.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. J. Gordon Wilson, 104 So. Michigan Ave., Chicago.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Luther C. Peter, Physicians Bldg., Philadelphia.
Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Hotel Statler, St. Louis. Time: Oct. 15-19, 1928.

LOCAL

THE COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. Howard Darrow, Metropolitan Bldg., Denver.
Secretary: Dr. Harold L. Hickey, Imperial Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

SECTION ON OPHTHALMOLOGY AND OTO-LARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

Chairman: Dr. Walter A. Wells, The Rochambeau, Washington, D. C.
Secretary: Dr. J. N. Greear, Jr., 1740 M St., Washington, D. C.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Arthur J. Fort, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Arthur M. Corwin, 25 E. Washington St., Chicago.
Secretary: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Grill Room, Stevens Restaurant, 16 N. Wabash Ave., Time: 6 p. m., first Monday of each month from October to May.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Thomas Hood, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. Raymond Hume, Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital, New Orleans.
Time: 8 p. m., third Thursday of each month from September to June.

SECTION ON OTOLOGY AND LARYNGOLOGY OF THE BALTIMORE CITY MEDICAL SOCIETY

Chairman: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.
Secretary: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Ray Connor, Stroh Bldg., Detroit.
Secretary-Treasurer: Dr. William Fowler, Maccabees Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

THE KANSAS CITY EYE, EAR, NOSE AND THROAT SOCIETY

President: T. S. Blakesley, Lathrop Bldg., Kansas City, Mo.
Secretary: Dr. A. E. Eubank, 636 Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May; the November, January and March meetings are devoted to clinical work.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Gordon Berry, 19 Elm St., Worcester, Mass.
Secretary-Treasurer: Dr. Calvin B. Faunce, 320 Commonwealth Ave., Boston.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Henry C. Barkhorn, 45 Johnson Ave., Newark, N. J.
Secretary: Dr. Lee W. Hughes, 1019 Broad St., Newark, N. J.
Place: 91 Lincoln Park, South. Time: 8:45 p. m., second Monday of each month.

THE SECTION ON LARYNGOLOGY AND RHINOLOGY OF THE NEW YORK ACADEMY OF MEDICINE

Secretary: Dr. Arthur Nilsen, 40 E. 41st St., New York.
Time: 8:30 p. m., fourth Wednesday of each month from October to May.

THE OTOLOGICAL SECTION OF THE NEW YORK ACADEMY OF MEDICINE

Chairman: Dr. J. L. Maybaum, 12 E. 86th St., New York.
Secretary: Dr. R. T. Atkins, 4 W. 53d St., New York.
Place: 103d St. and Fifth Ave. Time: Second Friday of each month from October to May.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. Ivimey Dowling, 116 Washington Ave., Albany, N. Y.
Secretary-Treasurer: Dr. L. Prescott Brown, 33 Bancker St., Albany, N. Y.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Walter Murphy, 707 Race St., Cincinnati.
President-Elect: Dr. Charles C. Jones, 19 Garfield Pl., Cincinnati.
Secretary-Treasurer: Dr. Harris H. Vail, 24 E. 8th St., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

THE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SECTION OF THE CLEVELAND ACADEMY OF MEDICINE

Chairman: Dr. W. J. Abbott, 8314 Euclid Ave., Cleveland.
Secretary: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. W. J. Mellinger, Santa Barbara, Calif.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Santa Barbara, Calif. Time: April 18-20, 1928.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 13th & Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

THE SECTION ON OTOLOGY AND LARYNGOLOGY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

Chairman: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
Clerk: Dr. Curtis C. Eves, 1910 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. B. Selingstein, 801 Union and Planters Bank Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

EYE, EAR NOSE AND THROAT SECTION OF THE HOUSTON ACADEMY OF MEDICINE

President: Dr. Ray K. Daily, Medical Arts Bldg., Houston, Texas.
Secretary: Dr. L. W. Raney, Post-Dispatch Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of the mouth from September to June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. H. L. Warwick, Medical Arts Bldg., Fort Worth.
Secretary: Dr. A. E. Jackson, Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6 p. m., first Friday of each month except August.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Charles J. Coffey, 221 Grand Ave., Milwaukee.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

News and Comment

CHANGE IN DATE OF EXAMINATION BY AMERICAN BOARD OF OTOLARYNGOLOGY

The examination originally scheduled for Friday, Oct. 12, 1928, will be held on Thursday, October 11, at the Manhattan Eye, Ear, Nose and Throat Hospital, New York.